Effects of d-amphetamine on Choice in a Rapid-Acquisition Concurrent-Chains Procedure.

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Table of Contents

List of Figures

General introduction:

Experiment 1:

List of Tables

Abstract

Choice behaviour is an important topic for research in behavioural analysis and behavioural pharmacology. The matching law and generalized matching law have provided a good description for choice behavior in many studies. The purpose of the present study was to examine how d-amphetamine affects sensitivity and rate of acquisition of pigeons' choice between delayed reinforcers in the concurrent-chains procedure. Two experiments are reported, both used 'rapid acquisition' concurrent-chain schedules in which one terminal link was fixed-interval (FI) 8 s while the other terminal link changed across sessions between FI 4 s and FI 16 s according to a pseudo-random binary sequence (PRBS). In Experiment 1, the reinforcer magnitudes were constant and equal, but there were two types of trials in Experiment 2 in which magnitudes for both terminal links were either small or large. After baseline training, varied doses (e.g. 0.3, 1.0, 1.7 and 3.0 mg/kg) of d-amphetamine were administered prior to drug testing sessions. The results found that increasing doses of d-amphetamine reduced the sensitivity to the immediacy ratio in the current session, but no evidence that within-session acquisition was affected in Experiment 1, and no evidence that absolute magnitude affected preference or resistance to change in Experiment 2. Overall, these results show that d-amphetamine reduces sensitivity to delay, and that the rapid-

acquisition concurrent-chains design can provide a useful procedure for testing drug effects on choice.

General introduction on behavioral choice:

Since the early 1960s, a large number of experiments on operant conditioning, published in journals such as the Journal of the Experimental Analysis of Behavior and Animal Learning and Behavior, have dealt with choice behavior. It is not surprising that choice has been an important research topic, because arguably, all behavior involves choice in all species (Mazur, 2002). For example, animals in the natural environment can either maintain their current behavior or change to another at any time. The consequences of some choices can have different results. An example of an insignificant result would be if a chimpanzee used its left or right paw to pick up a piece of food, whereas other choices, such as to flee or fight an opponent, can have important and irreversible consequences. From the examples described above it is clear that an understanding of how organisms make choices is essential to an understanding of behavior itself (Mazur, 2002). Therefore it is not surprising that within experimental and applied behavior analysis considerable research has been conducted in this area (Ono, 2004). The most common variables which determine choice behavior include: (1) The contingencies of reinforcement in the environment; (2) Ontogenetic factors derived from the contingencies that have previously acted on the organism's behavior; (3) and

Phylogenetic factors derived from evolutionary contingencies that have acted on the organism's species (Ono, 2004).

Matching law and generalized matching law:

The "matching law", which was discovered and proposed by Richard Herrnstein (1961), is a simple mathematical expression that describes choice behavior in many situations. Herrnstein (1961) used a pigeon chamber with two response keys, a red key on the left and a white key on the right, located a few inches apart on one wall. Each key was associated with its own variable-interval (VI) schedule of reinforcement and hold consistently through his experiment. For example, pigeons that pecked a left key were reinforced with VI 135s and peck a right key were reinforced with VI 270s schedule. This schedule, that is, two or more reinforcement schedules that are presented simultaneously, is called a concurrent schedule (shown in Figure 1; Mazur, 2002). This VI 135s VI 270s concurrent schedule was changed to the next condition only when the response allocation in daily experimental appeared stable and so on. Only data from the last few sessions in each condition were used for subsequent analysis. Herrnstein found that the percentage of responses directed toward one alternative was approximately equal to, or "matched," the percentage of reinforcers delivered by that alternative. This relationship can be expressed mathematically as:

$$\frac{B_1}{B_1 + B_2} = \frac{R_1}{R_1 + R_2}. \quad (1)$$

In Equation 1, known as the matching law, B_1 represents the rate of responding on one response alternative and B_2 represents the rate of responding on a second alternative. R_1 and R_2 represent the respective rates of reinforcement for those alternatives. Note that Equation 1 can be rearranged algebraically to form ratios:

$$\frac{B1}{B2} = \frac{R1}{R2} \quad \textbf{(1a)}$$

Although Herrnstein's (1961) original study found that response ratios tended to match the ratio of reinforcement rates delivered by the two schedules, later research showed that there are several types of deviations from perfect matching that could occur. For example, undermatching could occurr, that is, response ratios may be less extreme than the reinforcement ratios; or in other hand, overmatching could occurr, that is, more extreme than the reinforcement ratios, and subjects may exhibit a bias, which is preference for one of the two alternatives that is independent of the reinforcer ratio (Mazur, 2001). All of these deviations from matching can be described by the **generalized matching law** (Baum, 1974), which can be written as follows:

$$\frac{B_L}{B_R} = b\left(\frac{R_L}{R_R}\right)^a \quad (2)$$

Equation 2 is the power function form for the generalized matching law; however, the logarithmic transformation is typically used as follows:

$$\log \frac{B_L}{B_R} = a \log \frac{R_L}{R_R} + \log b \quad (3)$$

Equations 2 and 3 extend the matching law by including two parameters. The parameter a is termed sensitivity of reinforcement and measures the extent to which the response ratio changes with changes in the reinforcer ratio. Log b is called response bias and is a constant proportional preference, independent of the reinforcer ratio, toward one of the alternatives. In other words, bias means unaccounted for preference. It suggests that some other variable affecting preference has not been measured. If all the variables were measured and incorporated into the expression estimating reinforcement, there would be no bias. Therefore, this reflects no fault on the part of the organism, but only the experimenter's inability to measure or control all the independent variables (Baum, 1974). When the two alternatives are similar, any bias thought to be due to a color or position preference is termed inherent bias.

Many studies (e.g. Baum, 1974, 1979, 1982; Davison & Jenkins, 1985; Staddon, 1968; Davison & McCarthy, 1988, and Davison & Hunter, 1976) have demonstrated that the generalized matching law provides an accurate description of choice. The logarithmic form of the generalized matching law is the most convenient, because fitting a least squares regression line to the dependent variable as a function of the independent variable yields a straight line function with slope a and intercept log b (Baum, 1979).

The most common result in studies using a generalized matching law analysis is undermatching, that is, when slope a been found to be less than 1.0, frequently around 0.8 (Baum, 1979; Davison & McCarthy, 1988; Williams, 1988, and Davison & Hunter, 1976). Undermatching refers to systematic deviation from the matching relation in the direction of indifference (i.e., response allocation that is less extreme than the reinforcer allocation; Baum, 1974). In proposing Equations 2 and 3, Baum (1974b) found that there

are several factors might affect sensitivity, such as penalties for switching between alternatives and failure of discrimination between alternatives. Although undermatching occurs frequently, its origins remain a puzzle. However, the size of *a* has been suggested to be influenced by the discriminability of the schedules, which depends on the difference between the stimuli signaling the two concurrent-schedule components (Miller, Saunders, & Bourland, 1980). Miller et al.'s (1980) study showed that response allocation depended on the difference between the stimuli associated with the different component schedules in a single-key procedure and undermatching increased when stimulus disparity decreased. In other words, when the stimuli were made more similar, greater undermatching was obtained.

Davison and Jenkins (1985) suggested that undermatching might be explained by a failure of discrimination due to misallocation of reinforcers between alternatives. The failure of discrimination between the stimuli associated with the different alternatives would result in some of each alternative's reinforcers being misallocated to the other. Therefore, they proposed a contingency discriminability model, which attempted to explain undermatching (Davison & Jenkins, 1985). Their goal was to develop the contingency discriminability model into an account of schedule and stimulus control that was both wider in application and conceptually clearer that the generalized matching law. They argued that the parameter (*dr*) in their model is conceptually better than the parameter (*a*) in the generalized matching law because ''sensitivity to reinforcement'' (*a*) gives no real explanation for why undermatching might occur, whereas decreases in contingency discriminability (*dr*), or increases in confusability, could lead to poorer ''matching'' by the subject.

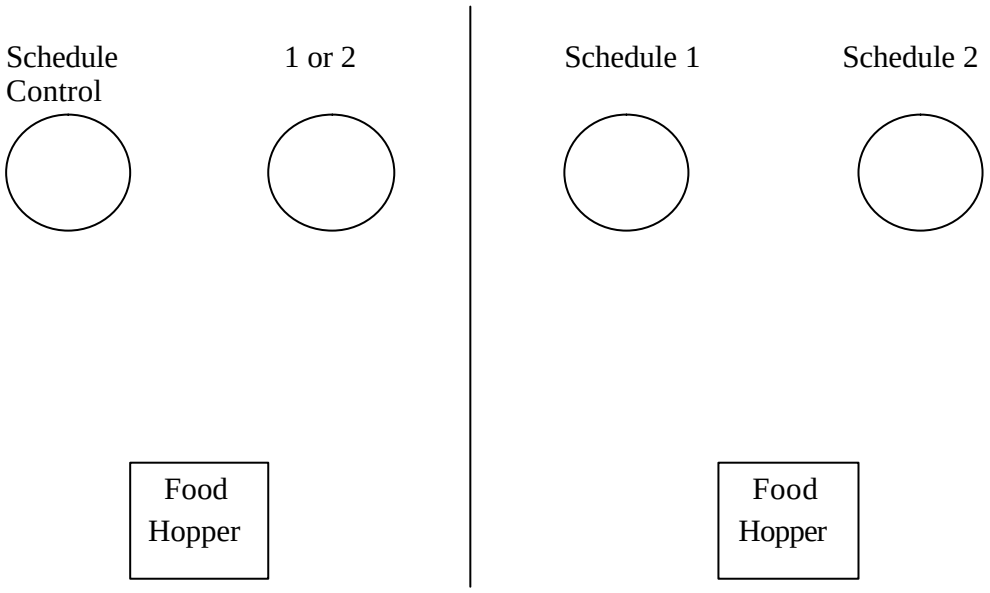


Figure 1: Representation of two methods of programming concurrent schedules. The left panel represents a changeover-key concurrent schedule, the right panel a two-key concurrent schedule.

Concurrent schedules vs. Concurrent-chain schedules:

Concurrent schedules (Figure 1) are the most common procedure used in research on behavioral choice. Concurrent schedules can be described as more than two schedules are arranged at the same time two or more operanda (e.g. levers, keys). All the

schedules are run sessions at the same time, and the subject is free to move between the operanda and schedules (Herrnstein, 1961). One schedule arranges a reinforcer and the other continues to time whether or not the arranged reinforcer has been taken is the independent scheduling arrangement for the concurrent schedules. Thus, it is possible that reinforcers for both responses may sometimes be available. By contrast, one schedule arranges a reinforcer and the other schedule stops timing until the arranged reinforcer has been taken is the dependent scheduling arrangement for concurrent schedules. The advantage of dependent scheduling is that the ratio of obtained reinforcers between the alternatives will be close (or equal) to the arranged ratio (Stubbs & Pliskoff, 1969). There are two measures of choice in concurrent schedule, response allocation, which is the ratio of responses emitted to both alternatives; and time allocation, which is ratio of time spent responding to each alternative. The changeover delay (COD), which is a short amount of time that must elapse following a switch between alternatives before a reinforcer can be delivered, also plays an important role in concurrent schedules (Herrnstein, 1961).

However, one limitation of concurrent schedules as a procedure for studying choice is that responding is reinforced directly with food. The generality of the matching law as a principle of behavior would be extended if it also applied to choice maintained by stimuli other than primary reinforcers. A Conditioned reinforcer, is an initially stimulus, for example, a red key light, has acquired the ability to serve as a reinforcer through being paired with a primary reinforcer (see Williams, 1994, for review). Therefore, a more complex procedure called concurrent-chains procedure (shown in Figure 2; Autor, 1960) arise and has been widely used in research on behavior choice. A

typical version of the procedure involves two response keys. Responses during an initial choice phase (initial links) are reinforced by access to one of two mutually-exclusive outcome schedules (terminal links). The onset of each terminal link is signaled with a distinctive stimulus (in Figure 2, green key for the left terminal link, red key for the right terminal link). Responding in either terminal link continues until food reinforcement is presented, and then the initial links are reinstated. The terminal-link stimuli are often called conditioned reinforcers because their ability to maintain initial link responding depends on a history of pairing with the primary reinforcer (e.g., food). According to this view, choice in the initial links measures the relative value of the terminal links as conditioned reinforcers (Grace, 2002).

Thus, the major difference between concurrent and concurrent-chains schedules is that in concurrent schedules, subjects can make one of two responses, each of which occasionally produces reinforcers. In concurrent chains, on the other hand, subjects choose between periods of access to reinforcement schedules signaled by distinctive stimuli. However, the concurrent chain procedures may be viewed as on a continuum, because shortening the terminal link reinforcement periods in concurrent chains moves the procedure closer to a concurrent schedule, and when the terminal links are 0 sec in duration, the procedure is a concurrent schedule (Davison & McCarthy, 1988).

Concurrent-chain schedules and the generalized matching law:

Davison and McCarthy (1988) showed throughout their book that the generalized matching law can act as a good descriptor and organizer of many of the data on choice in

concurrent schedules, but noted that it had not been applied so extensively to concurrent chain performances. The reason is that the generalized matching law can not explain many of effects that researchers have found, such as stimulus segmentation effects, the method of averaging terminal link reinforcer rates and the effects of the absolute durations of initial link schedules (Davison & McCarthy, 1988). However, Davison (1983) suggested that the concatenated generalized matching law, which was the extended from the generalized matching law, might apply in some situations to concurrent chains:

$$\frac{B_L}{B_R} = b\left(\frac{u_{1R}}{u_{1L}}\right)^{a1}\left(\frac{u_{2R}}{u_{2L}}\right)^{a2} \quad \textbf{(4)}$$

where b is bias, u_{1R} and u_{1L} are the mean initial link intervals between conditioned reinforcers, u_{2R} and u_{2L} are the mean terminal link delays to primary reinforcement and a_1 and a_2 are the sensitivities to relative initial link conditioned reinforcement and terminal link primary reinforcement rates. He found that the generalized matching law was useful in analyzing concurrent chain performance and that including a changeover delay in the initial links appeared to eliminate some interactions between initial and terminal links that have made quantitative predictions for concurrent chain performances difficult (Davison, 1983). However, Davison (1983) noted that Equation 4 could not explain effects of overall initial- and terminal-link duration on preference, and later compared the ability of several models to account for data from archival studies, and found that none of the models provided an adequate overall quantitative description of the data (Davison, 1987).

However, a subsequent model proposed by Grace (1994), called the contextual choice model (CCM), can provide an adequate quantitative description of wide range of concurrent chains data. CCM model is another extension the generalized matching law and it generalizes the generalized matching law through the incorporation of temporal context effects (Grace, 1994). The CCM model demonstrates that a matching law analysis of concurrent chains, i.e., the assumption that relative initial link responding equals relative terminal link value remains quantitatively viable (Grace, 1994). The contextual choice model can be written as:

$$\frac{B_L}{B_R} = b\left(\frac{u_{1R}}{u_{1L}}\right)^{a_1}\left(\frac{u_{2R}}{u_{2L}}\right)^{a_2\left(\frac{Tt}{Ti}\right)} \qquad \textbf{(5)}$$

where $\left(\frac{Tt}{Ti}\right)$ is the ratio of average time spent per reinforcement in the terminal links (Tt) to the average time spent per reinforcement in the initial links (Ti). The contextual choice model assumes that the crucial contextual variable in concurrent chains is the ratio of average times spent, per reinforcement, in the terminal and initial links; this ratio controls differential effectiveness of terminal link stimuli as conditioned reinforcers. Grace (1994) noted that CCM can account for the same qualitative effects as the delay reduction hypothesis (Fantino, 1969), with the additional advantage of accurate quantitative prediction across a wide range of schedules and procedural variations. Because the CCM model reduces to the generalized matching law when terminal link duration is zero also accounts for a high proportion of the variance overall in the archival data (ninety-two concurrent chains data sets; e.g. Davison, 1983, 1988; Wardlaw and Davison, 1974;

Davison and Temple, 1973), it provides a quantitative integration of concurrent schedules and concurrent chains.

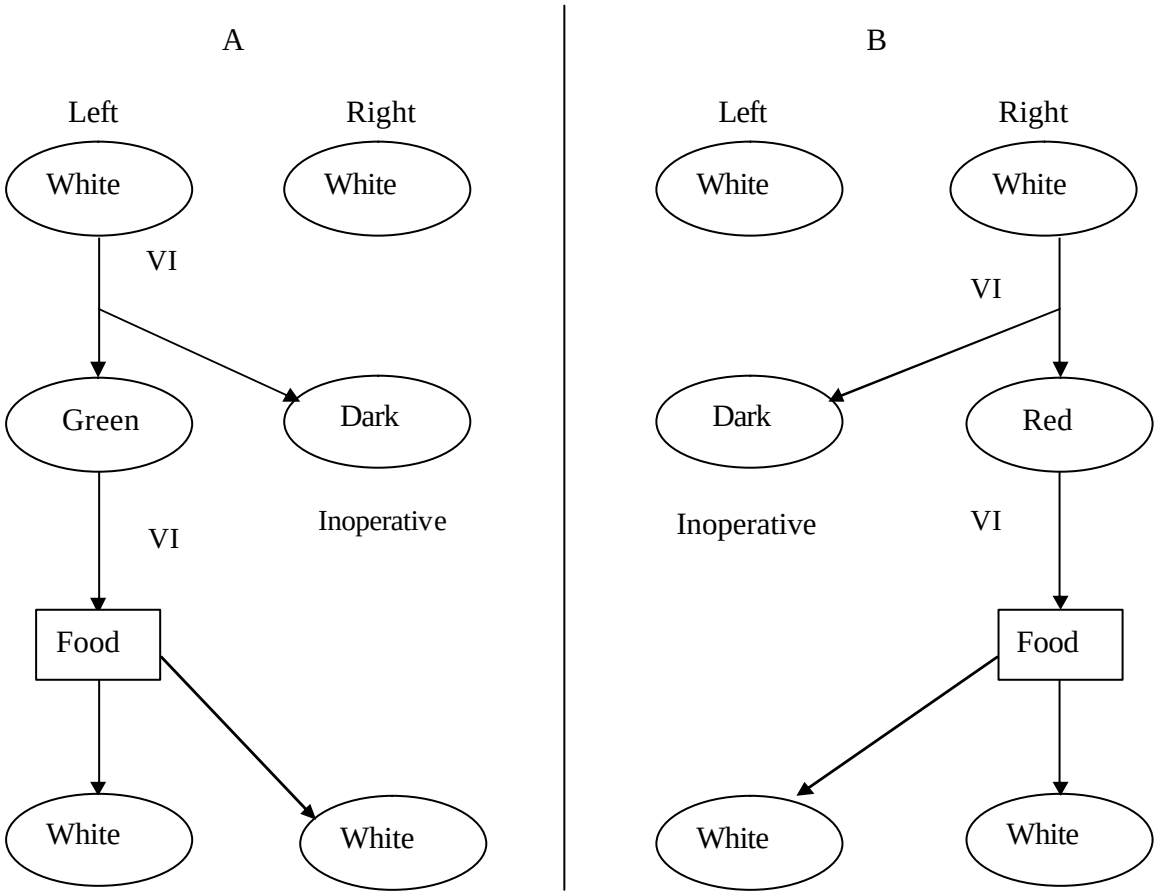


Figure 2: The concurrent-chains procedure. Panel A indicates the sequence of events when responses on the left key are reinforced; panel B presents the analogous sequence on the right key. Responses in the presence of the colored lights are reinforced with food

according to some schedule. The measure of choice is the relative rate of responding in the presence of the concurrently available white lights.

Acquisition or choice in transition:

Traditionally, research on choice has used "steady state" designs in which subjects have extensive experience with a particular set of reinforcement schedules. However recently, there have been a growing number of studies on acquisition of choice with either concurrent or concurrent-chain schedules (Grace, 2002; Grace, Bragason & McLean, 2003; Mazur, 1992, 1995; Mazur & Ratti, 1991; Mazur, Blake & McManus, 2001). For example, Grace, Bragason and McLean (2003) showed that pigeons' initial link response allocation can adjust rapidly to frequent changes in the terminal-link schedules. Using a successive-reversal design, Grace's (2002) study demonstrated that acquisition rate was faster when the terminal-link schedules preceding the reversal were FI and following the reversal were VI, compared to the opposite. Mazur and Ratti (1991) found that the two alternatives are more discriminable when the ratio of their reinforcement probabilities is larger and the acquisition of preference is faster. In Mazur, Blake and McManus (2001) found that the transition choice in animal is not only determined by the most recent change in the reinforcement contingencies, but also by the specific types of changes that have occurred in the animal's recent experience. Animals make choices on each of two response keys had the same probability of reinforcement, and subjects responded about equally often on the two key. However, when schedule values are shifted, that is, one key had a higher probability of reinforcement than the

other, the animals must make a new accurate prediction of reinforcement rate and the acquisition of preference for this key and the acquisition curve was observed (Mazur, 1992). The acquisition curve, or the rate of learning, declines systematically when experiment or practice proceeds is the old notion in psychology (Mazur & Hastie, 1978). Learning can be said to occur most rapidly in early training when requiring more and more practice in later training with equal increments in performance. From the practice result, these diminishing returns in learning curves are smooth, negatively accelerating functions (Mazur & Hastie, 1978). However, psychologists are relied on what is called "hyperbolic equations" and "hyperbolic acquisition curve (shown in equation 6 and Figure 3) over those two decades, more than any others in attempting to describe and analyze the learning process. Hyperbolic equations applied to learning curves are of the following general form:

$$y = k\left(\frac{t}{t+R}\right) \qquad \textbf{(6)}$$

When y is some measure of learning, t is the amount of time or the number of time in training, k is the asymptote for learning and R determines the rate of approach to this asymptote (Mazur & Hastie, 1978).

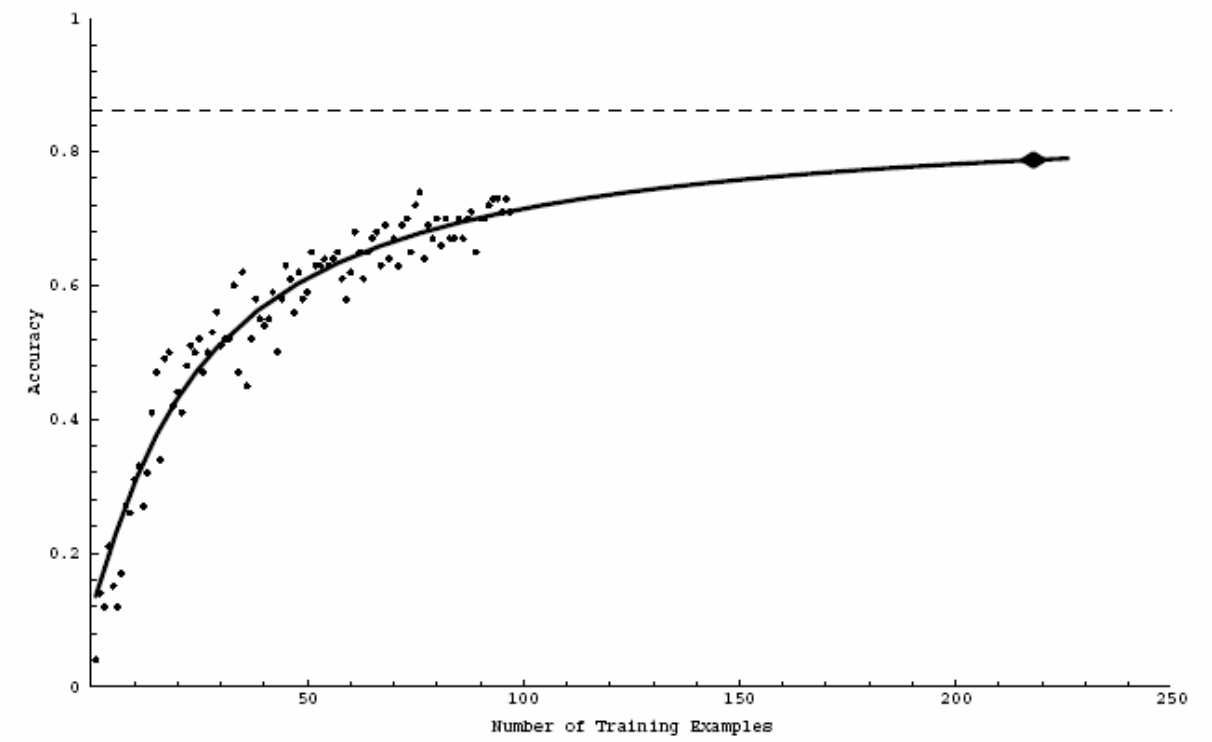


Figure 3: The thick curve shows the deterministic part of the learning-performance model (learning curve). The dotted line is the model's prediction of the largest achievable accuracy. The diamond shows that the actual accuracy achieved with examples is very close to the 78.8% that the model predicted.

PRBS design with concurrent schedule in acquisition:

Because of the larger number of growing interest in choice in transition, a "behavioral transfer function", that is, the dynamic effect of change in the reinforcer ratio within the sessions, was proposed by Hunter and Davison (1985) for pigeons' responding under concurrent VI VI schedules. Six pigeons were trained with either concurrent VI 60 s VI 240 s or concurrent VI 240 s VI 60 s in their experiment. They also proposed a powerful experimental method to conduct this experiment, that is, 31-step pseudorandom binary sequence (PRBS) (Hunter & Davison, 1985). In statistical terms, the PRBS

constituted "white noise", so that the reinforcer ratio for a particular session could not be predicted from the previous sessions' values. Davison and McCarthy's (1988) proposed an extension of the generalized matching law to analyze results from studies using PRBS designs:

$$\log \frac{B_{0L}}{B_{0R}} = a_0 \log \frac{R_{0L}}{R_{0R}} + a_1 \log \frac{R_{1L}}{R_{1R}} + a_2 \log \frac{R_{2L}}{R_{2R}} + \ldots\ldots\ldots + \log b \qquad \textbf{(7)}$$

This equation provides a way to describe the influence of the reinforcer ratio from the current and prior sessions on choice responding. In this equation, the log response ratio in a given session is determined additively by a series of terms representing the reinforcer ratio in the current and prior sessions. The sessions are indicated by the subscripts, with 0 for the current session and 1, 2… for the previous sessions. Each session has an associated sensitivity parameter (a_n). However, Davison and McCarthy found that sensitivity (a_0) for the current session (ie, Lag 0) was greatest and decreased rapidly to zero by approximately Lag 4 from this equation.

PRBS design with concurrent-chain schedule in acquisition:

A recent study by Grace, Bragason and McLean (2003) adapted the PRBS procedure by focusing on choice in transition in concurrent chains. In their experiment, terminal-link delays were changed unpredictably across sessions. In their experiment 1, a VI 10 s operated during the initial links, and ensured that the terminal links were entered equally often (i.e., dependent scheduling). In one of the terminal links, a red light signaled a fixed interval (FI) 8 s schedule and in the other terminal link, a green light signaled either an FI 4 s or an FI 16 s. The terminal link associated with the green light

changed across sessions according to the PRBS. Sessions ended after 72 initial and terminal link cycles had been completed. However, for a quantitative assessment of the degree to which the immediacy ratios from the current and previous sessions controlled choice, a generalized matching model similar to equation from Davison and McCarthy was applied to the data:

$$\log \frac{B_{0L}}{B_{0R}} = a_0 \log \frac{1/D_{0L}}{1/D_{0R}} + a_1 \log \frac{1/D_{1L}}{1/D_{1R}} + a_2 \log \frac{1/D_{2L}}{1/D_{2R}} + \ldots\ldots + \log b \qquad \textbf{(8)}$$

In Equation 8, B is initial link response rate and D is terminal link reinforcement delay, subscripted for both choice alternative (L and R) and Lag (0 thought 9). The parameters a_0 ….. a_9 represent sensitivity to reinforcement immediacy at each lag, and b is the bias. Multiple regression analyses were conducted to estimate sensitivity coefficients from Lag 0 (i.e., current session) through Lag 9. Separate analyses were conducted for the three PRBS presentations. Results showed that after sufficient (approximate 30 sessions) training with the procedure pigeons' response allocation during the initial links adjusted rapidly to the terminal link schedules at the start of each session, and pigeons was preferred the terminal link with the shorter delay to reinforcement at the end of the session. Specifically, Grace et al. found that responding became more sensitive to the immediacy ratio in the current session with increased training and the response allocation changed systematically during the first half of each session, but stabilized during the second half. In effect, their procedure yielded learning curves within each session.

Behavioral pharmacology:

The science of behavioral pharmacology is comparatively new and was been developed in recent years, even though behavior which affect by drugs have been studied widely for many years (Thompson & Schuster, 1968). However, most scientists recognized and agreed that behavior is a phenomenon amenable to study by the methods of the natural sciences (Thompson & Schuster, 1968). Ability to demonstrate precision and sensitivity of laboratory techniques developed for the experimental analysis of behavior are the reasons which make behavior pharmacology applicable in the social and biological sciences (Thompson & Schuster, 1968). Because of the efficacy of these techniques, behavioral pharmacology has been a rapidly developing discipline in the science of behavior.

Because many psychologists, pharmacologists and psychiatrists have been interested in the behavioral effect on drugs, the discipline has emerged as a science within the past 50 years. Most investigators found they need to increase their knowledge to know the methods and concepts of the related disciplines after they working at first in their various fields. In other words, with concerned drug-behavior interactions in both biological and social systems, behavioral pharmacology has become an interdisciplinary in science (Pickens, 1977). Over the past 40 years, the environmental contingencies controlling behavior have been repeatedly shown to play an important role in determining the behavioral effects of drugs in behavioral pharmacology (Branch, 1991). In other words, not only the behavioral actions of drugs can be understood in terms of biochemical or pharmacological principles, but also behavioral principles are important, that is, how behavior and environment influence in behavior and its events (Pitts & Febbo,

2004). Therefore, some behavioral pharmacologists have suggested that the notion of behavioral mechanisms of drug action could provide a useful framework for understanding how environmental contingencies modulate the behavioral effects of drugs (Pitts & Febbo, 2004; Branch, 1984, 1991; Thompson & Schuster, 1968; Thompson, 1984). The behavioral mechanism of drug action theory is based on the pharmacological notion of mechanism of drug action (Thompson & Schuster, 1968). From this point of view, a drug effect on a given biological system can be described when it can be shown that the effects were due to action of the drug on one or more of the basic mechanisms which control the system normally. Thus, the effect on behavior is explained when it is shown to be due to action of the drug on one or more variables which normally control behavior (Pickens, 1977). For example, there are various possibilities for why administration of a given drug changing the rate of schedule-controlled operant behavior; such as by changing the capacity to execute the response in organism; the effects of the establishing operation, the effects of the contingency between behavior and the consequent events, and the nature of the antecedent stimulus control (Pitts & Febbo, 2004). However, "rate-dependency theory", gives a different point of view as to how drugs affect behavior, proposed in recent years (Pickens, 1977). According to this view, drug effects depend on the baseline rate of the behavior emitted by the organism, with different rate-dependency functions for different classes of drugs (Kelleher & Morse, 1968). Overall, the most important and unifying theories developed by behavioral pharmacology are the behavioral mechanisms and rate-dependency theories; both of the theories can explain the behavioral effects of drugs. The difference between those two theories is behavior mechanisms theory seems more concerned with drug action on

variables that control conditioned behavior, whereas the rate-dependency theory is more concerned with motor output (Pickens, 1977).

Behavioral pharmacology and acquisition:

Recently, researchers have found that behavior pharmacology plays a very important role in acquisition or behavior in transition (Cohn & Paule, 1995; Thompson & Moerschbaecher, 1979). In general, behavioral pharmacology has focused primarily on the ability of drugs to affect performance of a well-learned behavior rather than on behavior in transition (Harvey, 1987). According to Laties (1979), an experiment which was carried out by I. V. Zavadskii in Pavlov's laboratory in 1908 is the original experiment that studied drug action on behavior in classical conditioning. In his experiment, Zavadskii studied the effects of alcohol, morphine, cocaine and caffeine on the conditioned salivary reflex in dogs (Laties, 1979). Therefore, many subsequent studies continued to use Pavlovian conditioning to investigate the effects of drugs on the performance of previously acquired responses. Arguably, Pavlovian conditioning provides the most accurate to determining the effects of drugs on learning (Harvey, 1987). For example, Gormezano et al. (1983) noted that Pavlov was the first person who succeeds antecedent injection procedure in behavioral effects of pharmacological drugs by using morphine to examined acquisition of the overt response in animals. They also mentioned that because of the conditioned stimulus verse conditioned response (CS-CR) procedure in classical conditioning will developed control methodology compared with

instrumental procedures, therefore, the classical conditioning appears to have a great deal of the behavioral effects with drug interventions (Gormezano, Kehoe and Marshall, 1983).

By contrast, the operant conditioning was also used to studies drug action on behavior. The first studies with operant conditioning were done by Dews in year 1955. Dews (1955) were set the stage for a technolo gy and paradigm in his experiment and this is became the predominant force in behavioral pharmacology. Therefore, most experiments in drug effects with behavior are used operant procedures in which the behaviors are maintained by a variety of events under various schedules of reinforcement (Harvey, 1987).

Amphetamine and acquisition:

The principal "tools" of pharmacology are drugs. A substantial literature has developed aimed at characterizing effects of stimulants, such as amphetamine, on acquisitio n of behavior in animals (e.g., Lesage, Byrne & Poling, 1996; Evans & Wenger, 1988; Paule & McMillan, 1984; Thompson, 1974; Cohn & Paule, 1995; Thompson & Moerschebaecher, 1979). From the viewpoint of the experiment al psychologist, the most interesting action of stimulants is the ability to facilitate goal-directed or operant behavior (Harvey, 1987). Amphetamines are powerful stimulants, known colloquially as "speed". The immediate effects of consuming such drugs are an increase in altertness and a decrease in feelings of fatigue and boredom (Julien, 2001). On the other hand, the fact that amphetamines can alter mood and increase self-confidence is the other major reason

for their use (Leavitt, 1974). Common stimulants include d-amphetamine (Dexedrine), methamphetamine (Desoxyn), dl-amphetamine (Adderall), and cocaine.

Moderate doses of amphetamine have been shown to be effective in reducing impulsiveness and hyperactivity in children diagnosed with attention deficit hyperactivity disorder (ADHD) (Gillberg et al, 1997; Findling and Dogin, 1998; Solanto, 1998). ADHD is one of the most common of the psychiatric disorders which appears in childhood. Children diagnosed with ADHD can't stay focused on a task, can't sit still, act without thinking, and have difficulty completing tasks. If untreated, the disorder can have long-term effects on a child's ability to make friends or do well at school or work. Over time, children with ADHD may develop depression, poor self-esteem, and other emotional problems. Approximately 2 million children in the United States had ADHD, that is, between 3 to 5 percent of children (Barkley, 2000; Wender; 2002; Wilens, 1999).

In non-clinical populations, researchers have found that moderate doses of amphetamine have beneficial effects on sustained performance and improve learning and memory (Rapoport et al. 1980; Soetens et al. 1995; Ward et al, 1997). In contrast, large doses of amphetamine (often taken by abusive users) have been associated with opposite behavioral effects, such as depression, feelings of paranoia, mood swings, panic attacks, anxiety problems, learning and memory impair and amphetamine-induced psychosis (Hall et al. 1996; Williamson et al. 1997; McKetin & Mattic, 1997, 1998; Snyder et al, 1974).

For effects of amphetamine on acquisition, Thompson and Moerschbaecher (1979) found that in their repeated-acquisition technique, errors increase and response rate decreased across various drugs (e.g. d-amphetamine, cocaine, imipramine, diazepam,

haloperidol and phencyclidine) and species (e.g. dogs, pigeons, humans, rats and monkeys) within the session. Other researchers also found that moderate doses of d-amphetamine increase the likelihood of choosing a larger, more delayed reinforcer (Richards et al, 1999). In other word, "self control" was enhanced by animals choosing larger, more delayed reinforcers or decreasing their response rate (sensitivity) to reinforcement delay after the moderate doses of amphetamine (Pitts & Febbo, 2004). For example, in Pitts and Febbo's (2004) experiment, the researchers were interested in the effects of a common stimulant drug (Methamphetamine) on self-control choices in pigeons. Pitts and Febbo used a concurrent-chains schedule to conduct their experiment. In the initial link, the white houselight and the side keys were illuminated, one was red and one was green. A single random interval (RI) 1 min schedule governed access to the terminal link. In the terminal link, fixed time (FT) schedules were used. One terminal link provided a small reinforcer (1s food) with signaled delay of 2s and the other provided a larger reinforcer (4s food) with signaled delay that varied from 2s to 40s within each session. In the baseline (no drug condition), preference for the larger reinforcer decreased as the delay increased from 2 s to 40 s. However, when given different doses (1.0 and 1.7 mg/kg) of methamphetamine, the pigeons increased their preference for the larger, relatively more delayed reinforcer (i.e., more self-control). In terms of behavioral mechanisms, this can be interpreted to mean that amphetamine increased the relative effectiveness of the larger food amount; in other word, amphetamine may have increased the sensitivity of the subjects' behavior to the difference in reinforcement amount. On the other hand, it also is possible that amphetamine attenuated the effects of reinforcement delay; that is, amphetamine may

have decreased the sensitivity of the subjects' behavior to the effects of delay (Richards et al., 1999).

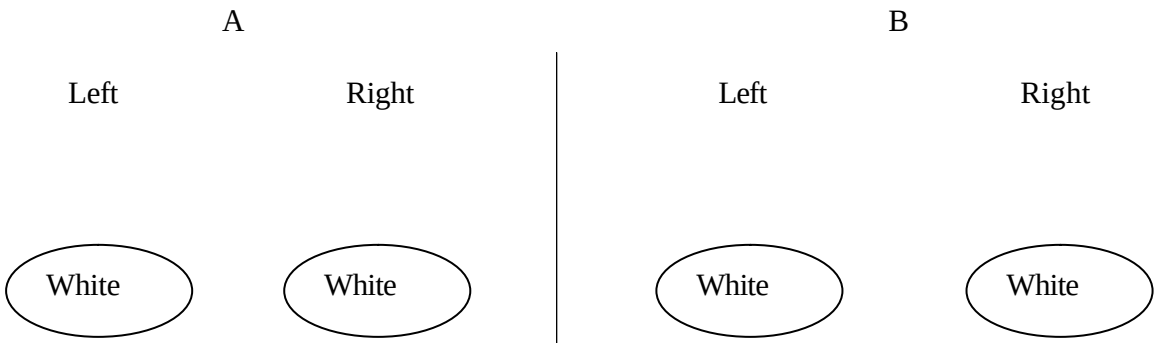
In addition, other researchers (Evenden & Ryan, 1996; Logue et al., 1992) found opposite effects, that is, moderate doses of d-amphetamine decreased the likelihood of choosing a larger, more delayed reinforcer (decrease self-control). However, Pitts and Febbo (2004) found that those studies in which self-control was increased after moderate amphetamine used explicitly signaled terminal-link delays, whereas those studies in which self-control was decreased after moderate amphetamine did not use explicitly signaled delays.

Present study:

There are two experiments in the present study. Experiment 1 will attempt to extend the PRBS concurrent-chains design from Grace et al (2003)'s Experiment 1 and use the procedure to explore the behavioral mechanisms of d-amphetamine in acquisition with pigeons (concurrent-chain diagram in present study shown in figure 4). There are two goals in experiment 1. The first is to determine whether pigeons will reduce their

sensitivity in delay after inject different doses (0.3 mg/kg, 1.0 mg/kg, 1.7 mg/kg, 3.0 mg/kg and 5.6 mg/kg) of d-amphetamine. The second goal is to analyze data within-session, to determine whether d-amphetamine affects rate of acquisition as well as steady-state preference.

Experiment 2 is similar as Experiment 1 in present study, the major difference being that sessions include two types of trials, in which the terminal-link reinforcers were either large or small reinforcer magnitude (4.5 s or 1.5 s access to grain, respectively). There are two goals in experiment 2. The first goal is to find out is there an effect of absolute magnitude on preference. The second goal is to test whether initial-link responding during the signalled large magnitude component would be more resistant to disruption than in the small-magnitude component. More details are described later in the introduction to Experiment 2.



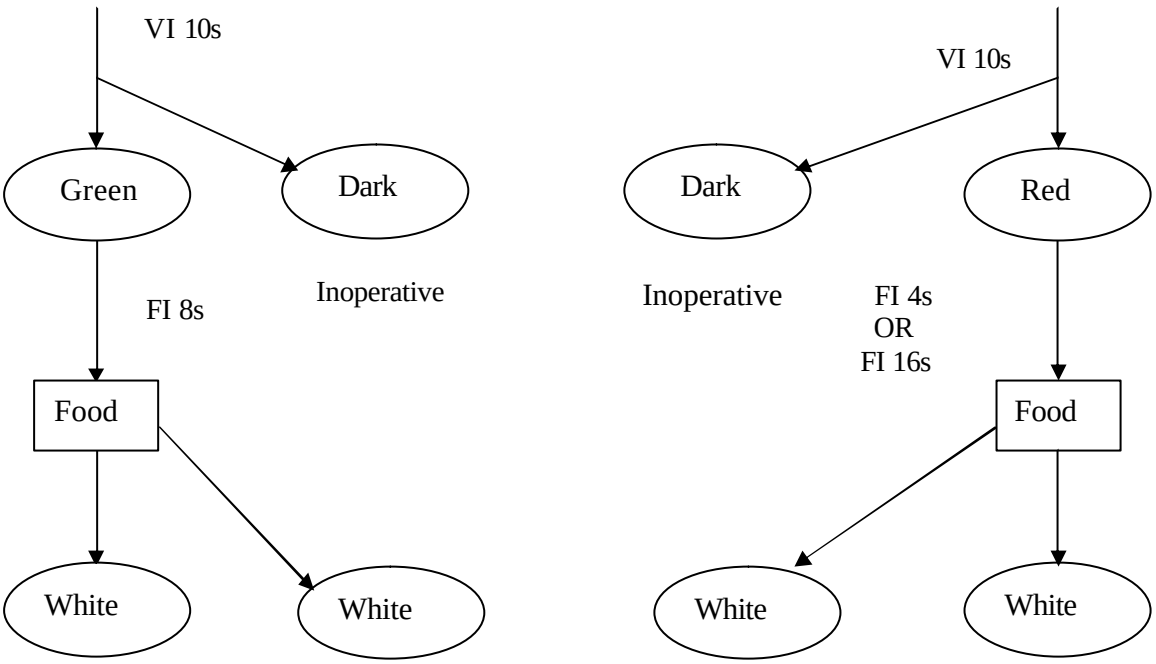


Figure 4: The concurrent-chain schedule for present study. VI 10s are the initial link for both Panel A and Panel B. FI 8s is the terminal links for Panel A and either FI 4s or FI 16s is the terminal links for Panel B (PRBS). Key-color position and the keys corresponding to the reinforcers were counterbalanced across pigeons and held constant throughout experiment.

Method

Subjects

Four pigeons of mixed breed, numbered 105, 106, 107 and 108, were maintained at 85% plus or minus 15g of their free-feeding weights through appropriate postsession feeding. Pigeons were housed individually in a vivarium with a 12:12 hr light / dark cycle (lights on at 6:00 a.m.), with water and grit freely available in the home cages. Pigeons were naïve at the beginning of this experiment and never experienced with a variety of experimental procedures before.

Apparatus

Four standard three-key operant chambers, 32 cm deep by 34 cm wide by 34 cm high, were used. The keys were 21 cm above the floor and arranged in a row 10 cm apart. In each chamber houselight that provided general illumination was located above the center key, and a grain magazine with a 5 by 5.5 cm aperture was centered 6 cm above the floor. The magazine contained wheat and was illuminated when wheat was made available. A force of approximately 0.15 N was necessary to operate each key. Each chamber was enclosed in a sound-attenuating box, and an attached fan provided ventilation and masking noise. Experimental events were controlled and data recorded through a microcomputer and MED-PC® interface located in an adjacent room.

Behavior procedure

The procedures for the present experiment are similar to those arranged in Experiment 1 of the Grace et al. (2003) paper. Because of the pigeons in present experiment do not have previous training experience, pigeons were adapted to the chambers and trained to eat from the food magazine. Autoshaping procedure was also used to train pigeons to pecking the colour keys (e.g. white, red or green) from the experiment chambers. The houselight provided general illumination at all times except during reinforcer delivery. With few exceptions, sessions occurred daily at approximately 10:00 a.m.

A concurrent-chains procedure was used in present experiment. Exposed pigeons to the concurrent VI 1 s schedules with white keys for one session before implementing the concurrent-chain procedure. Concurrent VI 10 s schedules were used over a couple of days after concurrent VI 1 s schedules then added FI 1 s terminal links for a day under concurrent VI 10 s schedules. After that, exposed pigeons to FI 4 s vs. FI 8 s in the terminal links and changed into FI 16 s vs. FI 8 s until pigeons are given the evidence of a preference for FI 4 s (shorter delays). After pigeons are shown enough evidence to prefer the shorter delays (e.g. prefer FI 4 s when the terminal links are FI 4 s vs. FI 8s; prefer FI 8s when the terminal links are FI 16 s vs. FI 8s), then implemented the PRBS procedure. There was a total of 74 sessions for Pigeons 105, 106 and 108; and 100 sessions for Pigeon 107 in the baseline condition. There are two training in the baseline condition, preliminary and PRBS. 33 sessions are the preliminary training for Pigeon 105, 106 and 108; 38 sessions for Pigeon 107. Preliminary sessions training was used either FI 8 s and FI 16 s or FI 16s and FI 8 s in the terminal link for all subjects. After all subjects developed strong preference in the short delay (e.g. 8 s), PRBS were conducted. There

are 41 sessions for PRBS training with Pigeons 105, 106 and 108; 62 sessions for Pigeon 107. The last 30 sessions in PRBS training were used for data analysis in present study. Each session ended after 72 initial and terminal-link cycles or 70 min, whichever occurred first.

At the beginning of a cycle, the side keys were lighted white to signal the initial links. An entry was assigned randomly by change of colour on key from white to red (left key) or white to green (right key) in terminal links coupled with the other key begin darkened. The initial links were VI 10-s schedules that guaranteed equal numbers of left and right terminal link entries (36 per session). A 1-s changeover delay (COD) was during the initial links as well. Thus, the first response to the pre-selected key after the VI 10 s schedule timed out produced a terminal link entry, provided the COD was satisfied.

The VI 10 s initial-link schedules did not begin timing until the first response had occurred in each cycle, to allow any pausing after the completion of the previous terminal link to be excluded from initial-link time. The VI 10 s schedule contained 12 intervals constructed from an exponential progression. Separate lists of intervals were maintained for cycles in which the left or right terminal link had been selected and were sampled without replacement so that all 12 intervals would be used three times for both the left and right terminal links each session (Grace et al, 2003).

Responding during the terminal link schedules was reinforced with access to grain according to FI schedules. Schedule values were changed across sessions according to the two independent 31-step PRBS sequences, which was the same as that used by Hunter and Davison (1985). In PRBS terminal link, one terminal link was always FI 8 s

(the standard delay) and the other was either FI 4 s or FI 16 s (the variable delay, specified in Table 1). The value of the variable delay was constant within a session, but changed across sessions according to the PRBS. Upon entry into a given terminal link, the colour of the designated side key changed from white to red or green, and the other side key was darkened. Keypositions and colours corresponding to the terminal links were counterbalanced across pigeons and held constant throughout the experiment. The conditions for each pigeons were as follows: 105, left key: either FI 4 s or FI 16 s / red, right key: FI 8 s constantly / green; 106, left key: FI 8 s constantly / green, right key: either FI 4 s or FI 16 s / red; 107, left key: either FI 4s or FI 16s / red, right key: FI 8 s constantly / green; 108, left key: FI 8 s constantly / green, right key: either FI 4 s or FI 16s / red. When a terminal link response was reinforced, the grain magazine raised and lighted for the specified 3 s duration, with access to grain. During the reinforcement, all illumination in the chamber was extinguished, and the grain magazine light was illuminated. After reinforcement, the initial links were reinstated.

	Step	**Delay**	**Step**	**Delay**

1	4	17	16
2	4	18	4
3	16	19	16
4	4	20	4
5	4	21	4
6	4	22	16
7	16	23	16
8	4	24	4
9	16	25	4
10	4	26	4
11	16	27	4
12	16	28	4
13	16	29	16
14	16	30	16
15	4	31	16
16	16		

Table 1: The delay of 31-step PRBS in the terminal links with FI schedule for the green key (Bird 105 and 107) or the red key (Bird 106 and 108).

Pharmacological procedure

There were two phases in the experiment, baseline and drug testing. During baseline, subjects received sessions as described above. Once pigeons' responding stabilized in the baseline, drug testing began during the second PRBS. During the drug test phase, *d*-amphetamine was dissolved in saline (sodium chloride) and injected 15 min prior to select sessions. Injections were given into the breast muscle (i.m.), usually in a volume of 1.0 ml/kg. Occasionally, the volume of a higher concentration was adjusted downward to achieve a desire dose. Injections were administered once or twice week, provided that the data from the session conducted the day before (the control session) were within the range of the previous 10 non-injection sessions; if this was not the case, the injection for that day was cancelled, but the session still was conducted (Pitts &

Febbo, 2004). Effects of the saline vehicle were determined at least twice prior to the initiation of drug testing. All sessions preceded by injections were separated by a minimum of two days. The doses of d-amphetamine were used in present study were: 0.3, 1.0, 1.7, 3.0, and 5.6 mg/kg (shown in Table 2). Doses were administered in a mixed order with the constraint that no dose was given a second time until all doses had been given once. However, each dose was given in pairs, once at FI 4 s and once at FI 16 s before testing the next dose. Once each dose had been tested in pair, doses of 1.0 and 1.7 mg/kg are the probed in additional times.

Pigeon 105	**Pigeon 106**
Saline--- Three doses for FI 4 s; Two doses for FI 16 s. 0.3 mg/kg--- Two doses for FI 4 s; Two doses for FI 16 s. 1.0 mg/kg--- Two doses for FI 4s; Two doses for FI 16 s. 1.7 mg/kg--- Two doses for FI 4 s; Two doses for FI 16 s. 3.0 mg/kg---- One dose for FI 4 s; One dose for FI 16 s.	Saline--- Two doses for FI 4 s; Two doses for FI 16s. 0.3 mg/kg--- Two doses for FI 4 s; Two doses for FI 16s. 1.0 mg/kg--- Two doses for FI 4 s; Three doses for FI16 s. 1.7 mg/kg--- Two doses for FI 4 s; Three doses for FI16 s. 3.0 mg/kg--- One dose for FI 4 s; One dose for FI 16 s.
Pigeon 107	**Pigeon 108**
Saline--- Two doses for FI 4 s; Three doses for FI 16 s. 0.3 mg/kg--- Two doses for FI 4 s; Two doses for FI 16 s. 1.0 mg/kg--- Two doses for FI 4 s; Two doses for FI 16 s. 1.7 mg/kg--- Two doses for FI 4 s; Two doses for FI 16 s. 3.0 mg/kg--- One doses for FI 4s.	Saline--- Three doses for FI 4 s; Two doses for FI 16 s. 0.3 mg/kg--- One dose for FI 4 s; One dose for FI 16 s. 1.0 mg/kg-- Two doses for FI 4 s; Two doses for FI 16s. 1.7 mg/kg---Two doses for FI 4 s; Three doses for FI 16 s. 3.0 mg/kg--- Two doses for FI 4 s; One dose for FI 16 s. 5.6 mg/kg--- One dose for FI 4 s.

Table 2: Doses of the drug administration (d-amphetamine) were used for Pigeons 105 through 108 in present study.

Results and Discussion

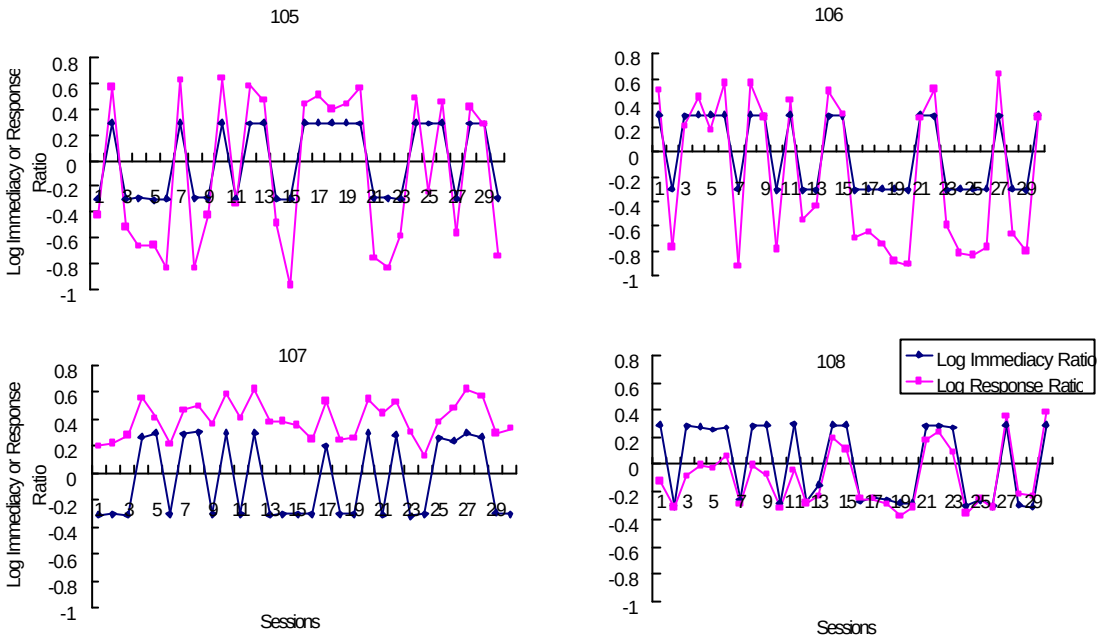


Figure 5: Log immediacy and Log response ratios across the last 30 days before the first drug injection (baseline condition).

Baseline

To characterize performance in the pre-drug baseline, a series of analyses was conducted on the data from the last 30 sessions prior to the first drug administration. Figure 5 shows the log immediacy and log response ratios for the last 30 baseline sessions before the first drug injection. Each pair of data points represents performance in a single session. The filled diamonds indicate the log immediacy ratio or delay ratio, that is, the logarithm of the delay on the right divided by delay on the left (e.g. 4s/ 8s). The filled squares indicate the log response ratio, that is, the logarithm of the responses on the left key divided by the responses on the right key. From the Figure 5 we can see that response allocation for pigeons 105 and 106 track the log immediacy very well across all 30 sessions – these pigeons consistently made relatively more responses to the

initial link associated with the shorter terminal-link delay. Thus, response allocation for Pigeons 105 and 106 shows strong sensitivity to the immediacy ratio. Response allocation for Pigeon 107 does not track the log immediacy as well as Pigeons 105 and 106, but still shows some sensitivity to the immediacy ratio. In addition, Pigeon 107 seems to have a strong left-key bias across all 30 sessions. Response allocation for Pigeon 108 did not track the log immediacy ratio over the first 12 sessions in Figure 5, but tracked well after the 13th session. This subject also appears to have a right-key bias during the first 12 sessions. Overall, data in Figure 1 shows that all subjects were able to track daily changes in the immediacy ratio, similar to Grace et al (2003).

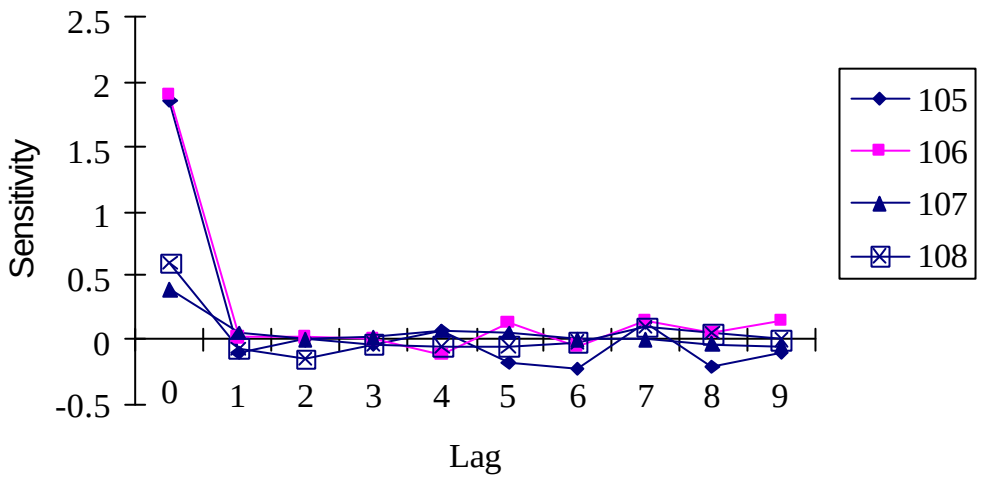


Figure 6: Overall sensitivity to immediacy from Lag 0 through Lag 9 for Pigeons 105, 106, 107 and 108.

Figure 6 shows results from a multiple regression analysis of the data from the last 30 baseline sessions before the first injection. This analysis used a generalized-matching

model with sensitivity coefficients for both the current-session immediacy ratio and the immediacy ratios from prior sessions (i.e., Lag 0 through Lag 9):

$$\log \frac{B_{0L}}{B_{0R}} = a_0 \log \frac{1/D_{0L}}{1/D_{0R}} + a_1 \log \frac{1/D_{1L}}{1/D_{1R}} + a_2 \log \frac{1/D_{2L}}{1/D_{2R}} + \ldots\ldots + \log b \,. \quad (8)$$

Equation 8 allows for a quantitative assessment of the degree to which the immediacy ratios from the current and previous sessions controlled response allocation in baseline. All subjects are accounting for between 72% to 97% variance in present study for overall regression from Equation 8. e.g. Pigeon 105, 93%; Pigeon 106, 97%; Pigeon 107, 72% and Pigeon 108, 81%. Overall sensitivity to immediacy in regression coefficients for Pigeon 105 shows a significant result for Lag 0 (sensitivity=1.84, $p<0.05$) whereas coefficients for Lag 1 through Lag 9 were never significant (varied from -0.09 to -0.10). Pigeon 106 shows sensitivity coefficients were significant for Lag 0 (sensitivity=1.90, $p<0.05$) whereas coefficients for Lag 1 through Lag 9 were never significant (varied from 0.02 to 0.15). Pigeon 107 shows sensitivity coefficients were significant for Lag 0 (sensitivity=0.39, $p<0.05$) whereas coefficients for Lag 1 through Lag 9 were never significant (varied from 0.05 to -0.04). Pigeon 108 shows sensitivity coefficients were significant for Lag 0 (sensitivity=0.60, $p<0.05$) whereas coefficients for Lag 1 through Lag 9 were never significant (varied from -0.06 to 0.00). Compared with Grace et al.'s (2003) experiment, response allocation for pigeons in present study shows higher overall sensitivity to the Lag 0 immediacy ratio (comparable values ranged from 1.84 to 0.47 for Grace et al, 2003). Thus, Figure 2 shows that there were individual

differences in sensitivity, but that response allocation for all pigeons was sensitive to the immediacy ratio in the current session.

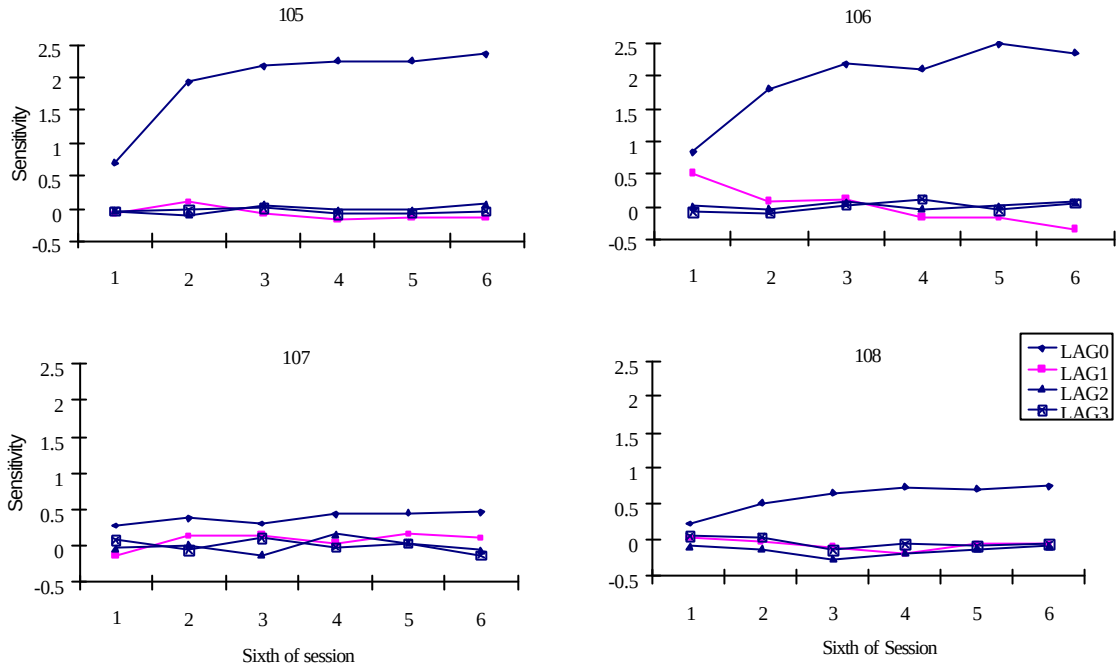


Figure 7: Sensitivity to immediacy for Lag 0 through Lag 3 determined separately for each session sixth.

Figure 7 shows results of the multiple regression analysis for the 30 days before first drug injection, conducted separately for data from successive sixths (i.e., 12 reinforcer blocks) of each session. The purpose of this analysis was to characterize how sensitivity coefficients changed within sessions. For all subjects, Lag 0 sensitivity increased over the course of the session, and sensitivities either decreased or varied unsystematically around zero for Lag 1 through Lag 3. For Pigeons 105, 107 and 108, Lag 0 sensitivity coefficients were always positive and statistically significant, whereas coefficients for Lag 1 through Lag 3 were never significant. This indicates that response allocation for Pigeons 105, 107 and 108 was controlled by the current-session immediacy ratio, with

little or no influence of prior sessions. For Pigeon 106, in addition to Lag 0 coefficients, Lag 1 was positive and statistically significant for the first of the sessions. These significant results for Lag 1 mean that there was some control by the prior session for Pigeon 106, but this control diminished over the course of the session. Overall, Figure 7 shows that the present study found similar results as Grace et al (2003), that is, an increasing Lag 0 sensitivity within the session for all subjects. Moreover, Lag 0 sensitivity increased over the first half of the session and did not show further changes across the second half of the session for all pigeons, also replicating Grace et al (2003).

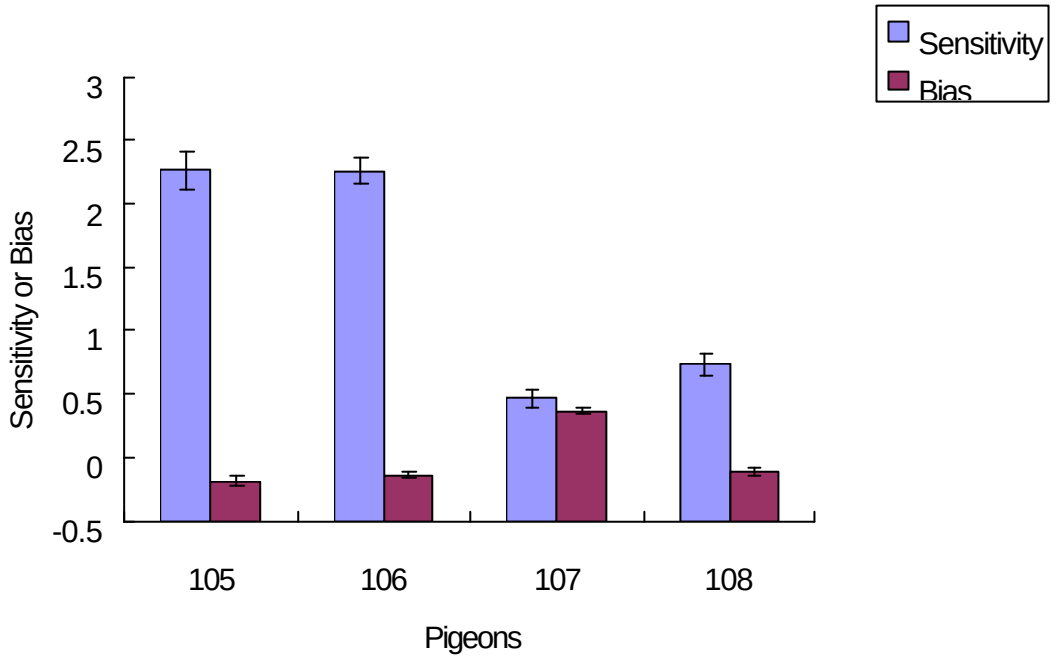


Figure 8: Sensitivity and bias for Pigeons 105, 106, 107 and 108 in second half of the session (4^{th}, 5^{th} and 6^{th}). Error bars indicate plus or minus one standard error.

Because the Lag 0 sensitivity values stabilized over the second half of the session in Figure 8, similar to Grace et al. (2003), to summarize asymptotic performance we conducted regression analyses on data pooled over the second half of the sessions.

Figure 4 shows the resulting sensitivity (Lag 0) and bias coefficients for all subjects. Sensitivity is relatively high for both Pigeons 105 and 106 (2.26 for both Pigeons), and is lower, by contrast, for both Pigeons 107 and 108 (0.47 for Pigeon 107 and 0.73 for Pigeon 108). In Figure 4, we can also see that Pigeons 105, 106 and 108 have right-key bias (less than 0) and Pigeon 107 has left-key bias (great than 0) in the second half of the session.

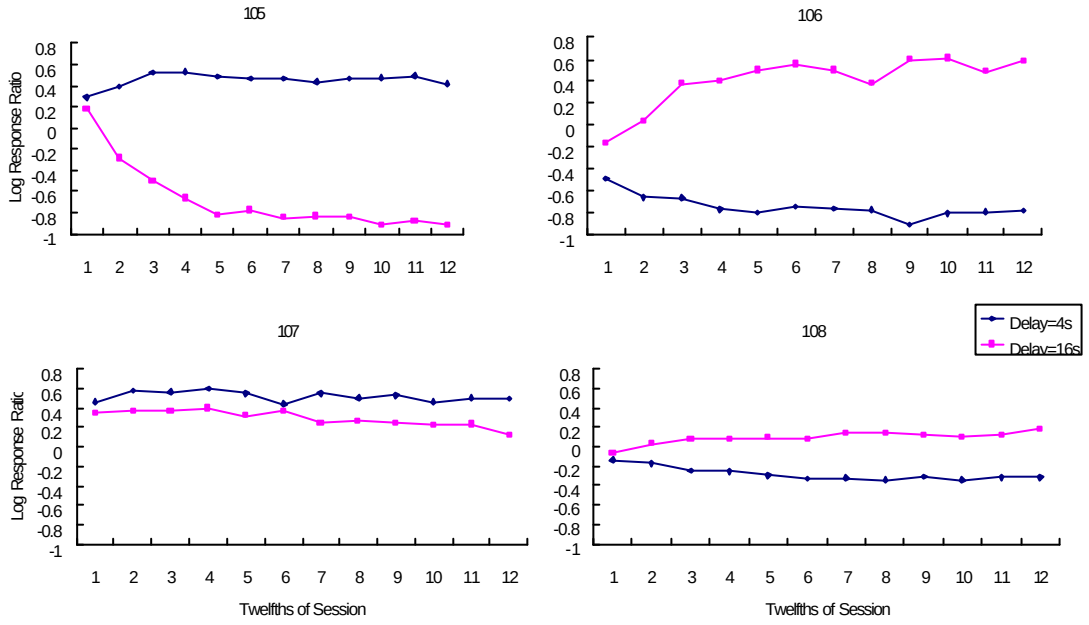


Figure 9: Log initial link response ratio (left/right) for each session twelfth. Two separate lines indicate whether the green key FI schedule delay value was either 4s or 16s. Note that the FI 8-s terminal link was associated with the right initial link for Pigeons 105 and 107, and with the left initial link for Pigeons 106 and 108.

To examine within-session changes in response allocation in more detail, Figure 9 shows the log initial-link response ratio for successive twelfths of the sessions. Data were tabulated separately for sessions in which the green-key FI schedule was FI 4 s or FI 16 s. The diamond shape indicates the 4s delay value or green key in FI schedule. The

top diamond shape line for Pigeons 105 and 107 indicates 4s delay on right key and the bottom diamond shape line for Pigeons 106 and 108 indicates 4s delay on left key. The square shape indicates the 16s delay value or green key in FI schedule. The top square shape line for Pigeons 106 and 108 indicates 16s delay on right key and bottom shape line for Pigeons 105 and 107 indicates 16s delay on left key. Pigeon 105 showed a slight left key bias, which is associated with green terminal link and Pigeons 106 and 108 showed right key biases, which is associated with green terminal link and strong left key bias for Pigeon 107 which is associated with green terminal link.

The difference between the first and twelfth data points in Figure 9 can be used to measure the extent of change for the 4s and 16s green key sessions. These differences were calculated as follows for Pigeon 105: 0.13 for 4s and 1.10 for 16s; Pigeon 106: 0.30 for 4s and 0.75 for 16s; Pigeon 107: 0.03 for 4s and 0.21 for 16s; Pigeon 108: 0.18 for 4s and 0.24 for 16s. This result shows that for all subjects, the extent of change for the 16s delay green key sessions was greater than for the 4s delay green key sessions. This indicates that the absolute value of the log immediacy ratio may not have been the sole variable controlling choice, as that was the same for both 4-s and 16-s green-key delay sessions. The larger within-session change for the FI 8s and FI 16s schedules compared to FI 8s and FI 4s schedules is similar to the "terminal link effect" in concurrent chains, in which preference between a pair of schedules in constant ratio (2:1) becomes more extreme as their absolute duration increases (Grace & Bragason, 2004; MacEwen, 1972; Williams & Fantino, 1978).

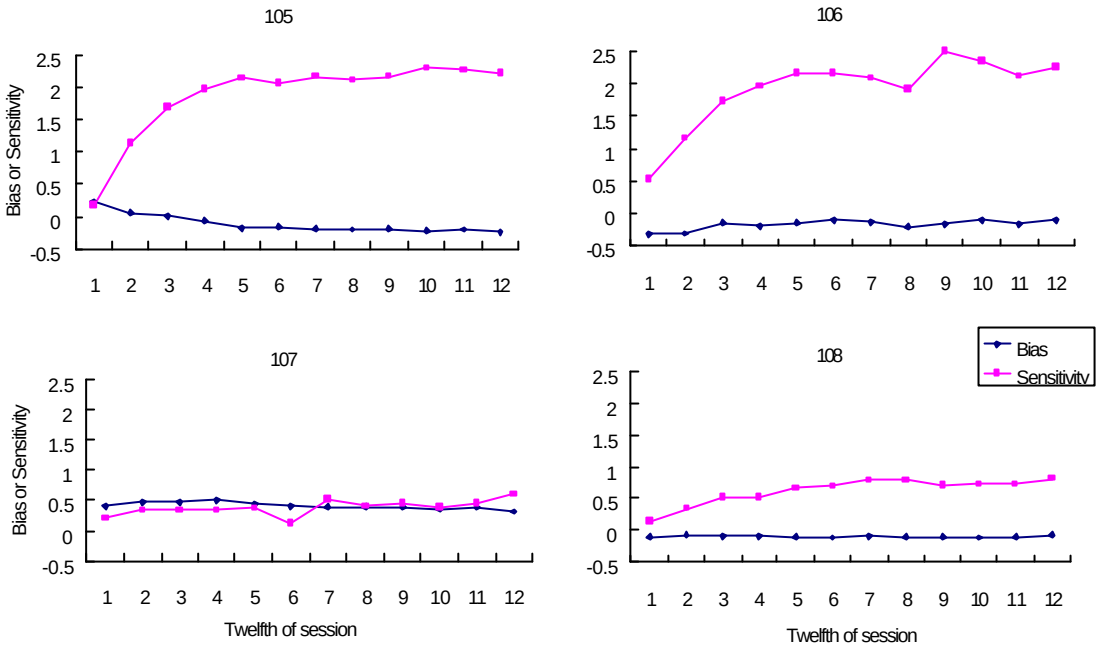


Figure 10: Bias and sensitivity in baseline condition for Pigeons 105 to 108 in twelfth of session.

Another way of examining within-session changes in preference is provided by Figure 10, which shows bias and sensitivity for successive session twelfths in the baseline condition. Basically, Figure 10 presents the same data as in Figure 5, that is, the log response ratio in left or right key for sessions in which the delay is 4 s, for each 12th of the session, and correspondingly for sessions in which the delay is 16 s, except re-expressed as bias and sensitivity. Bias was calculated as the average of the two data points (i.e., log response ratios when the alternative terminal link was either FI 4s or FI 16s), and sensitivity was calculated as the difference between the log response ratios divided by 2*Log(2). In effect, this approach calculates point estimates of slope (sensitivity) and intercept (bias) when a generalized-matching model is fit to the pair of data points from each twelfth of the session (i.e., FI 8 s/FI 4 s and FI 8 s/FI 16 s).

Figure 10 shows that sensitivity increased across the session for all subjects, however, once again pigeons 105 and 106 have higher sensitivity than other two pigeons. Pigeons 106 and 108 show a slight right-key bias, pigeon 105 shows a slight left-key bias, and pigeon 107 shows a strong left-key bias in Figure 6. Compared with Figure 8, which is based on the second half of the session (4^{th}, 5^{th} and 6^{th} sixths), Figure 10 shows similar results, that is, high sensitivity for Pigeons 105 and 106 and lower sensitivity for Pigeons 107 and 108; Expect Pigeon 105, which developed left key bias, Pigeons 106, 107 and 108 are all show the same results between Figure 8 and 10.

Summary of Baseline Results

Overall, results from the last 30 baseline sessions show that response allocation in the initial link can be sensitive to unpredictable changes in terminal-link FI schedules across sessions (Figure 5). There were individual differences in sensitivity, particularly sensitivity to the immediacy ratio in the current session (Figure 6). Response allocation was controlled primarily by the current session immediacy ratio (Figure 7), and relatively high sensitivity values were obtained in the second half of the session for all subjects (Figure 8 and Figure 10). All subjects also have "terminal link effects" in that the increase in preference within-sessions for the shorter delay was greater when the alternative terminal link was FI 16 s than when it was FI 4 s. (Figure 9). These results are similar to what Grace et al's (2003) study found; however, the sensitivity and bias for all subjects in present study are higher than Grace et al (2003) study. In addition, the sensitivities for present study are higher than Grace et al's (2003) study can be depended

on total number of sessions training. For Grace et al's (2003) study, all pigeons had 93 sessions of PRBS training. The sensitivity was found increase with longer training. However, the total number of sessions training in baseline for present study (74 in total) was less than Grace et al's (2003) study but found higher sensitivity than Grace et al's (2003). This indicated that increase sensitivity does not necessary need longer training.

Drug Testing

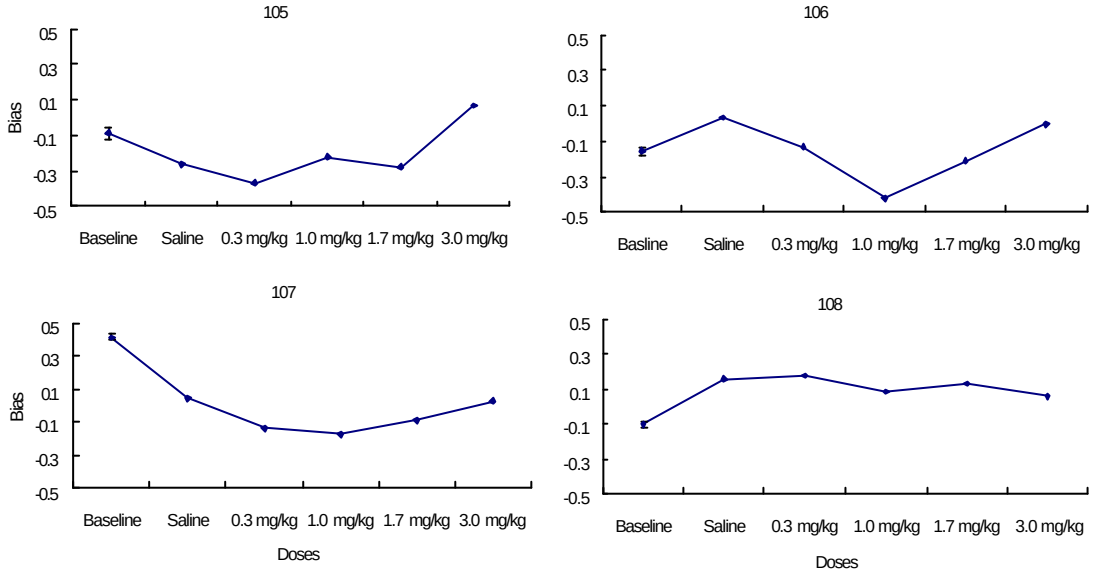


Figure 11: Bias and different doses of d-amphetamine compared with overall baseline condition. Error bars indicates plus and minus one standard error for overall baseline.

Several analyses to elucidate the effects of amphetamine on response allocation were conducted. For these analyses, data from test sessions with the same dose (or saline) and delays were pooled.

Figure 11 shows how bias changed during the test sessions compared with overall baseline condition. Across pigeons, there were no systematic changes in bias in the test

sessions. Pigeon 105 showed almost no bias in baseline but developed a slight left key bias at 3.0 mg/kg. Pigeon 106 is shown strong right key bias at 0.3 and 1.0 mg/kg but eliminated after 1.7 and 3.0 mg/kg. Pigeon 107 showed a strong left key bias in baseline, but this was eliminated with increasing doses of d-amphetamine. Pigeon 108 showed a right key bias in baseline, but shifted towards a slight left key bias as the amphetamine dose increased. Pigeon 108 failed to respond at all with dose of 5.6 mg/kg, therefore, this condition was omitted from the analyses. Overall, there were no systematic changes for bias with different doses of d-amphetamine.

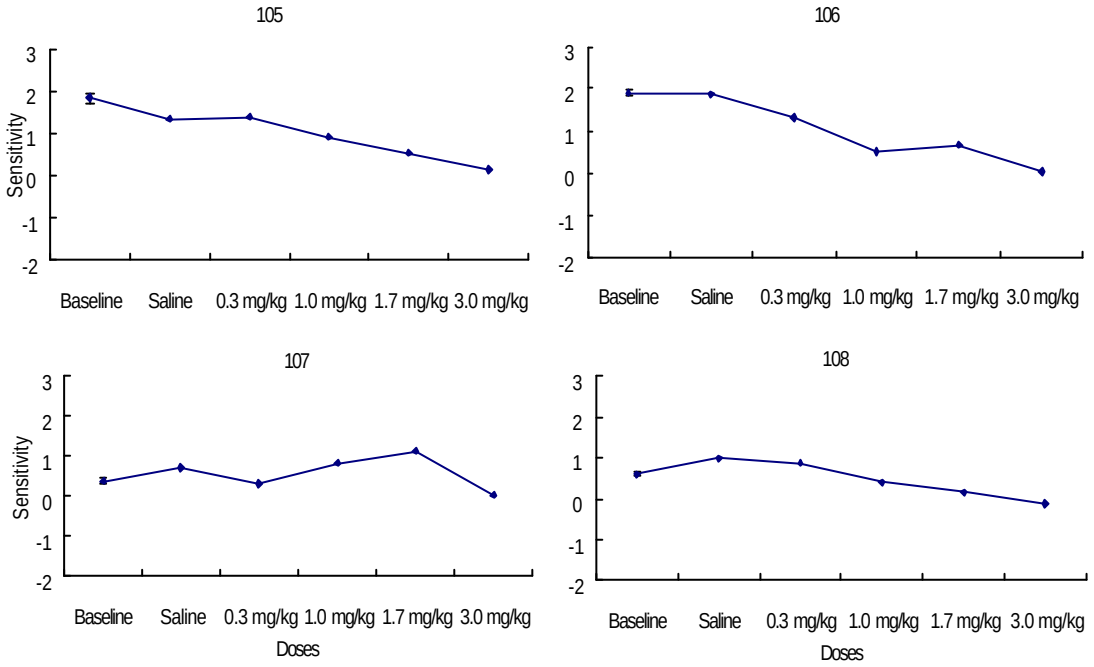


Figure 12: Sensitivity and different doses of d-amphetamine compared with overall baseline condition. Error bars indicates plus and minus of one standard error for overall baseline.

Figure 12 shows how sensitivity (Lag0) to the immediacy ratio changed in the test sessions compared with baseline. Pigeons 105, 106 and 108 showed the decreasing

sensitivity when given doses of d-amphetamine is increased. However, Pigeon107 showed increasing sensitivity when given doses of d-amphetamine is increased, but sensitivity decreasing when given the higher does of d-amphetamine (3.0 mg/kg).. Overall, Figure 8 shows clearly results that sensitivity decreased as the doses of d-amphetamine increased for Pigeons 105, 106 and 108. Opposite effects for Pigeon 107, that is increasing sensitivity as the doses of d-amphetamine increased but decreasing when given does of 3.0 mg/kg. Pitts and Febbo (2004) found that sensitivity will decreased when given the larger doses of amphetamine. Pigeons 105, 106 and 108 are the closed results to confirm what Pitts and Febbo (2004).

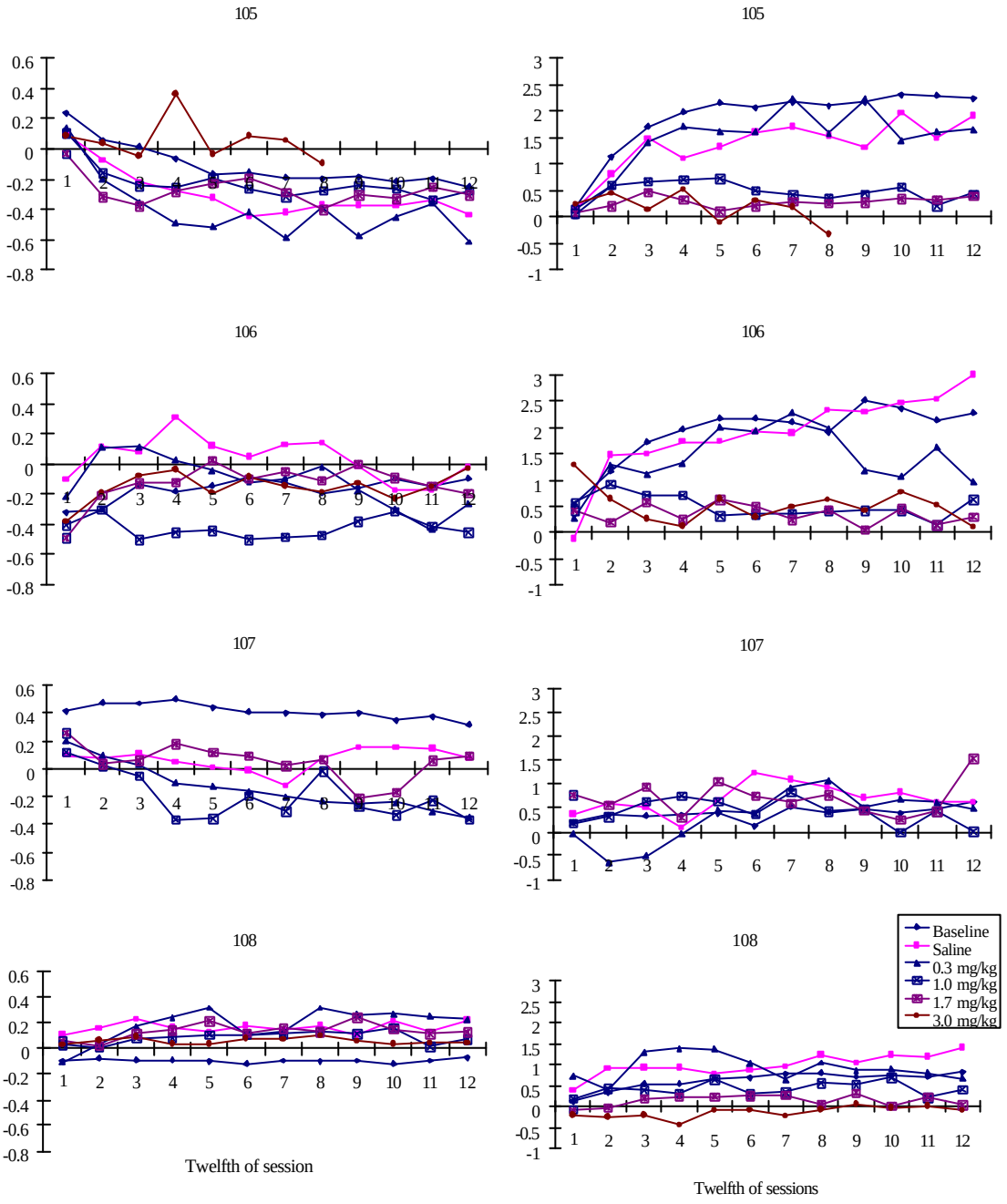


Figure 13: Bias (left panels) and Sensitivity (right panels) for different doses of d-amphetamine for each session twelfth. Data from baseline and saline conditions are also shown.

To investigate how amphetamine affected acquisition of response allocation within sessions, data from test sessions were analyzed according to each successive session twelfth, as in Figures 9 and 10. Figure 13 shows bias (left panels) and Lag 0 sensitivity (right panels) for each session twelfth and for each dose of d-amphetamine. Data from baseline and saline conditions are also shown for sake of comparison. For Pigeon 105, bias decreased in saline, 0.3 mg/kg, 1.0 mg/kg and 1.7 mg/kg doses of d-amphetamine; however, response virtually ceased at doses of 3.0 mg/kg. For Pigeon 106, bias increased in saline, 0.3 mg/kg and 1.7 mg/kg doses of d-amphetamine, relative to baseline. However, dose of 1.0 mg/kg of d-amphetamine produced a decreased bias, and again there was little responding at 3.0 mg/kg doses. For Pigeon 107, very obviously, bias decreased for all doses of d-amphetamine; the decreases in order were: 1.7 mg/kg, saline, 0.3 mg/kg and 1.0 mg/kg. Contrary to Pigeon 107, Pigeon 108 showed clearly increasing bias for all doses of d-amphetamine; the increases in order were: 3.0mg/kg, 1.0 mg/kg, 1.7 mg/kg, saline and 0.3 mg/kg.

Corresponding values for Lag 0 sensitivity are presented in the right panels of Figure 13. For Pigeon 105, sensitivities decreased as the dose of d-amphetamine were increased. Similar results were obtained for Pigeon 106: sensitivities decreased in order from 0.3 mg/kg, 1.0 mg/kg, 1.7 mg/kg and 3.0 mg/kg. For Pigeon 107, the results are not as clear as Pigeons 105 and 106, that is, shown the increased patterns in sensitivity when doses are increased; only the sensitivity decreased in dose of 0.3 mg/kg in the beginning of the twelfth of session. The increased sensitivities in order are: 0.3 mg/kg, saline, 1.0 mg/kg and 1.7 mg/kg. For Pigeon 108, shown similar results as Pigeon 105, that is, the sensitivities decreased at the higher doses of 1.0 mg/kg, 1.7 mg/kg and 3.0 mg/kg, but did

not decrease (or slightly increased) patterns for saline and 0.3 mg/kg. The decreased sensitivities in order are: 1.0 mg/kg, 1.7 mg/kg and 3.0 mg/kg. In summary, no systemically change in bias with different doses of d-amphetamine for all subjects. Pigeon 105, 106 and 108 confirm expectations based on the prior literature, that is, sensitivity to delay will decrease when given large doses of d-amphetamine.

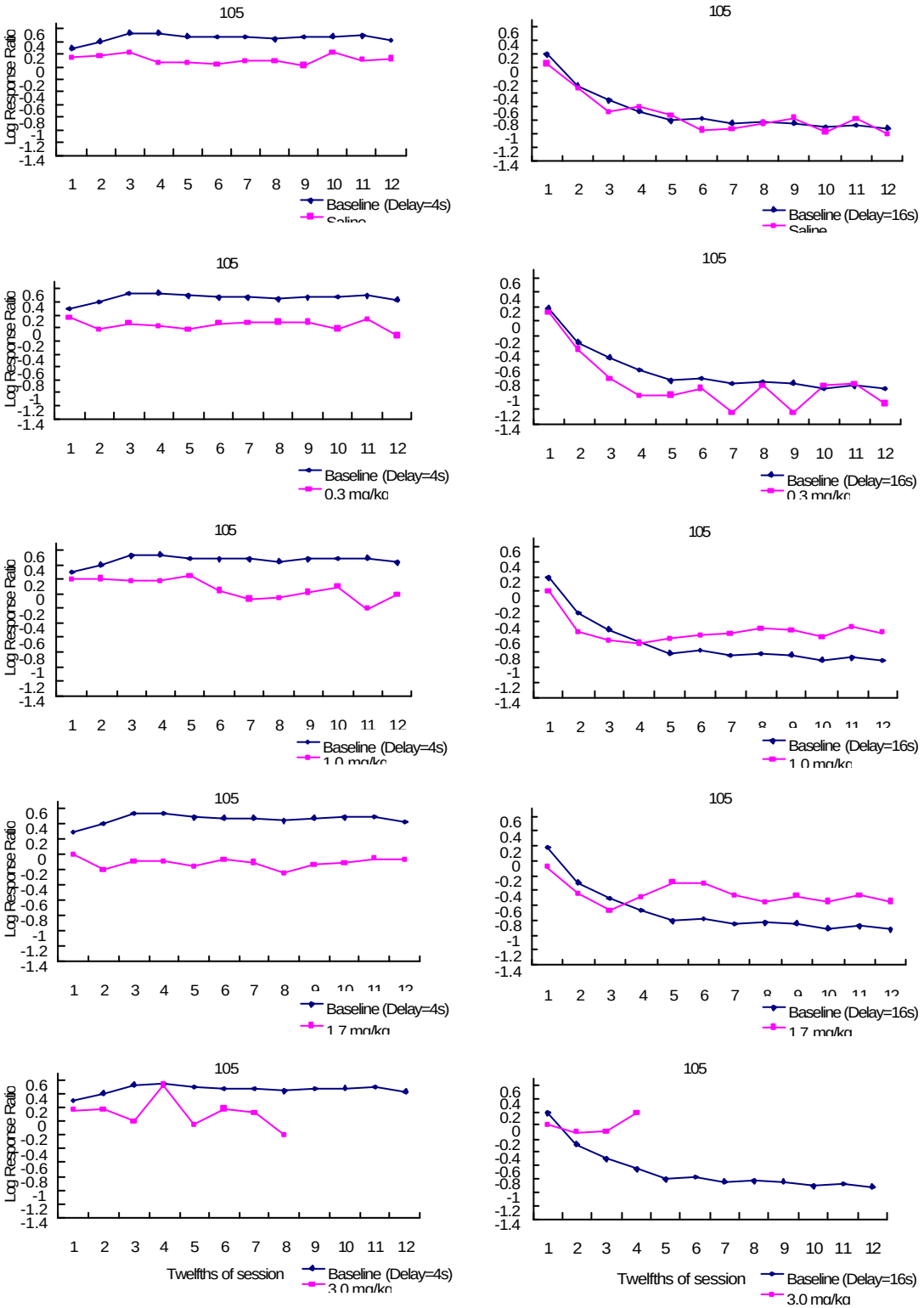


Figure 14: Log response ratio in delay 4 s and delay 16 s with each doses of d-amphetamine in twelfth of sessions for Pigeon 105.

Figure 14 shows log response ratios for Pigeon 105, separately for alternative delay values of FI 4 s and FI 16 s, for each dose of d-amphetamine and session twelfth. The reason to analyze the data in this way is to investigate whether doses of d-amphetamine affected response allocation differently for 4 s and 16 s alternative delay sessions. In the saline condition compared with baseline, Pigeon 105 showed a decreased pattern for the 4 s delay but no obvious change in the 16 s delay. When given doses of 0.3 mg/kg d-amphetamine, both the 4 s and 16 s delays showed a decreased pattern (i.e., decreasing towards indifference). However, 4 s delay shown more decreased pattern than 16 s delay. When given doses of 1.0 mg/kg d-amphetamine, response allocation with both delays showed a decreasing pattern from first through the fourth session twelfth, but increased pattern after fourth session. Similar results were found with doses of 1.7 mg/kg compared with 1.0 mg/kg d-amphetamine. When given doses of 3.0 mg/kg d-amphetamine, 4 s delay showed a decreased pattern (expect the fourth session twelfth), and similar results were obtained for the 16 s delay, but responding ceased after the fourth session twelfth. Overall, preference for Pigeon 105 decreased towards indifference for both 4-s and 16-s delay sessions.

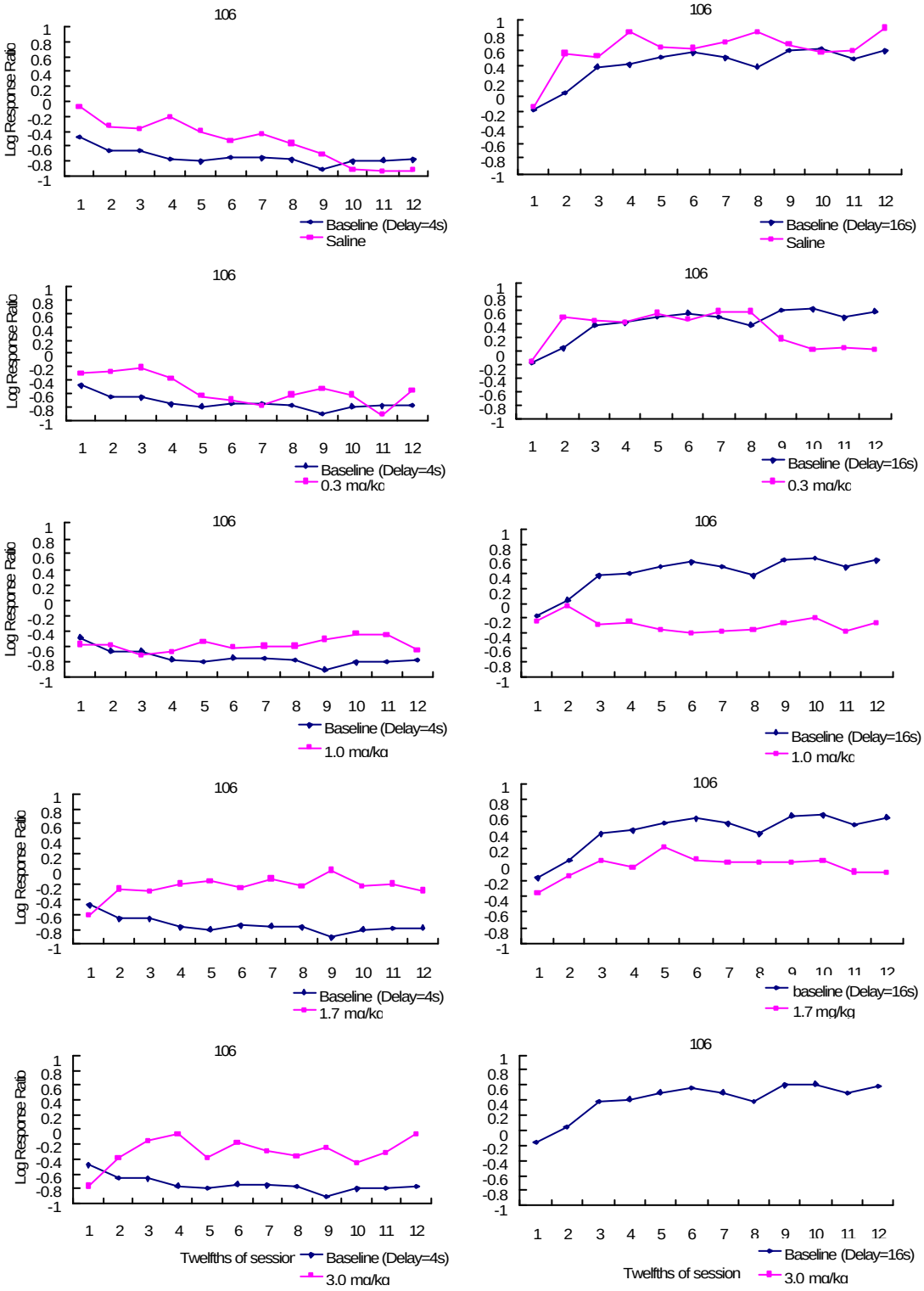


Figure 15: Log response ratio in delay 4 s and delay 16 s with each doses of d-amphetamine in twelfth of sessions for Pigeon 106.

Figure 15 shows the log response ratio increasing almost in every doses of d-amphetamine for delay of 4 s. However, the log response ratios decreasing when increase the doses of d-amphetamine for delay of 16 s for Pigeon 106. Overall, Pigeon 106 shows increased pattern in log response ratio when each doses of d-amphetamine for 4 s delays and opposite results found for 16 s delays.

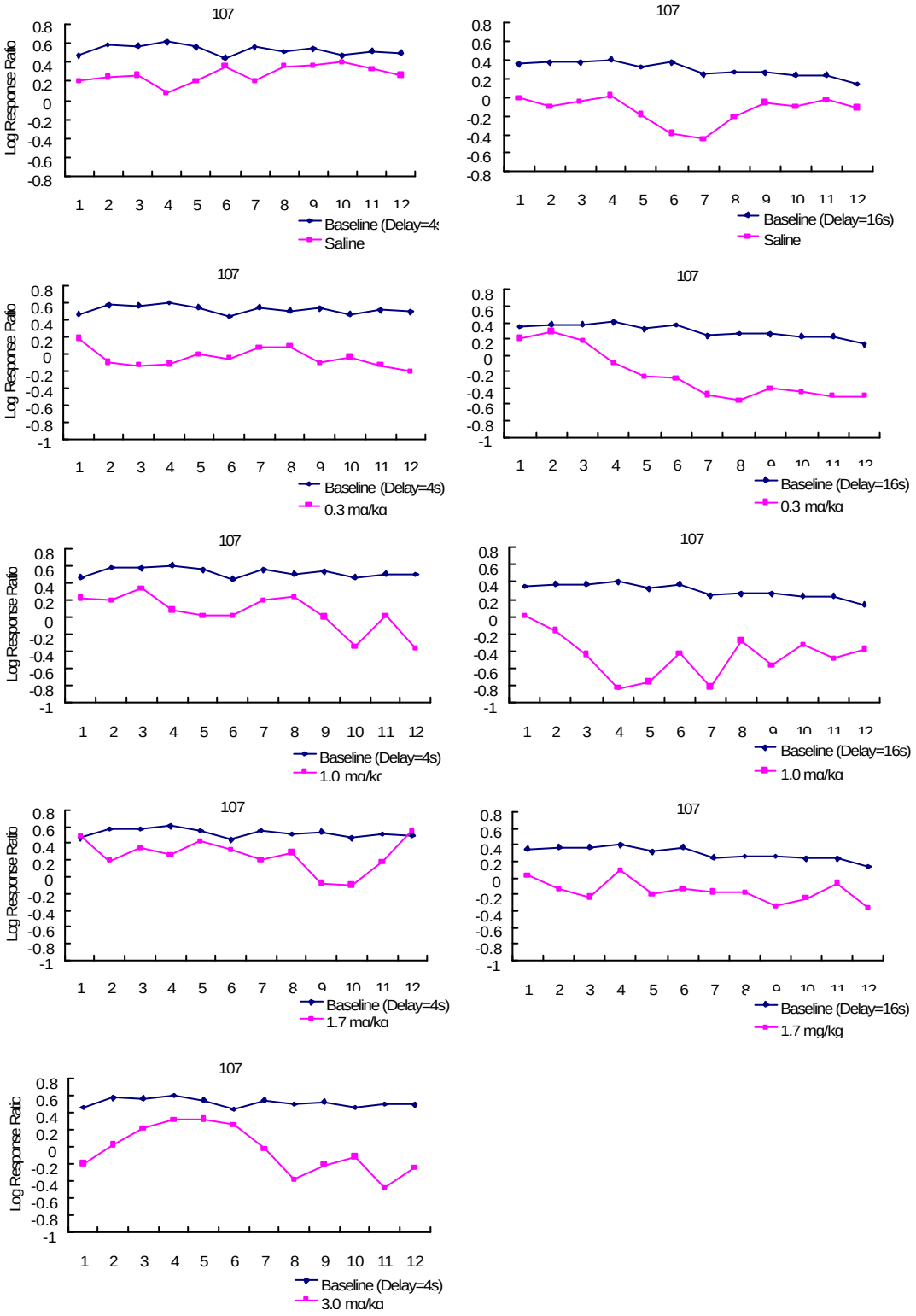


Figure 16: Log response ratio in delay 4 s and delay 16 s with each doses of d-amphetamine in twelfth of sessions for Pigeon 107.

Figure 16 is shown for Pigeon 107 with delay of 4 s and 16 s in each doses of d-amphetamine within the session. Except after tenth of session in doses of 1.7 mg/kg with delay of 4 s, Log response ratio was decreased in rest of doses (saline condition, doses of 0.3, 1.0, 1.7 and 3.0) for both 4 s and 16 s delays. 16 s delays are shown decreased more than 4 s delays in overall results.

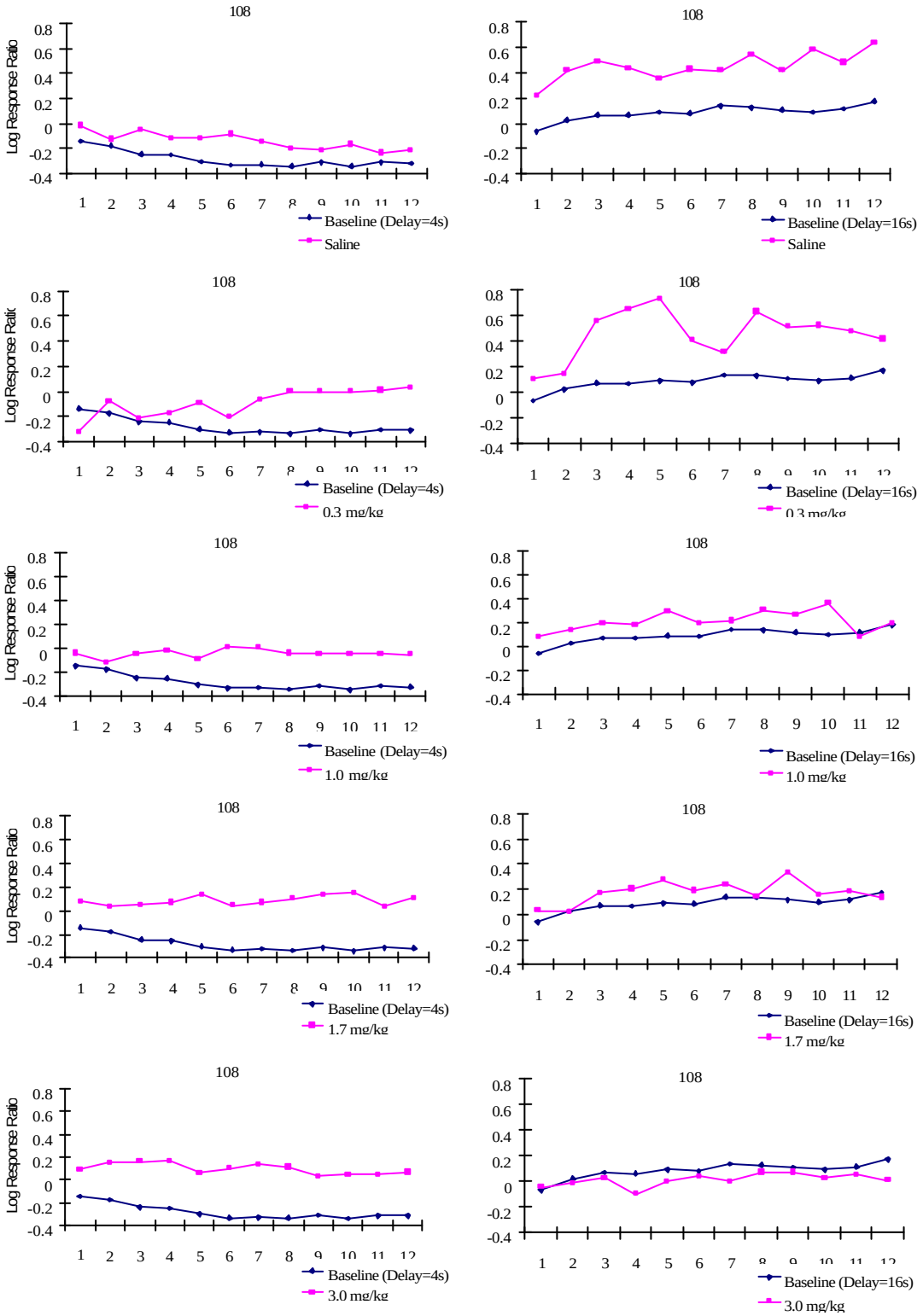


Figure 17: Log response ratio in delay 4 s and delay 16 s with each doses of d-amphetamine in twelfth of sessions for Pigeon 108.

Corresponding results from Pigeon 108 are shown in Figure 17. Log response ratio was increased in saline condition, doses of 0.3, 1.0 and 1.7 mg/kg for both 4 s and 16 s delays. Doses of 3.0 was shown increased in 4 s delay but decreased in 16 s delay.

Overall, Figure 14 to Figure 17 showed different changes for Log response ratio with different delay value and different doses of d-amphetamine in each subject. Pigeons 105 and 107 showed decreased Log response ratio s when the delay was 4 s with each doses of d-amphetamine; Pigeons 106 and 108 are shown increased in Log response ratio when the delay was 4 s with each doses of d-amphetamine. Except Pigeon 105, rests of pigeons are shown decreased in Log response ratio when increase doses of d-amphetamine when the delay was 16 s. On average, Log response ratio are shifted more in 16 s delays rather then 4 s delays for all subjects.

Summary for drug results:

Across subjects, there were no systematic changes in bias when doses of d-amphetamine was increased (Figure 11). Pigeon 105 is shown sensitivity increased and Pigeons 106 and 108 are shown sensitivity decreased when given doses of d-amphetamine was increased; Pigeon 107 is given mixed results (Figure 12). When look at the results in twelfth of sessions, except Pigeon 107, all subjects are shown sensitivity decreased and bias increased when given increased doses of d-amphetamine (Figure 13). All subjects are shown different changes for Log response ratio with different delay value and different doses of d-amphetamine in each subject (Figure 14 to 17). Figure 14 to 17

also shown that in average, Log response ratio are changes more in 16 s delays rather then 4 s delays for all subjects.

Experiment 2

Introduction:

Experiment 2 was a replication of Experiment 1, except that two types of concurrent-chains cycles were included, in which the reinforcers were either both small magnitude (1.5 s access to food) or large magnitude (4.5 s access to food). Similar to Experiment 1, the initial-link schedule was a VI 10 s that arranged equal entries into the terminal links, which were FI 4 s/FI 16 s on the green key and FI 8 s on the red key. Trial-types were signalled by presence (or absence) of a flashing houselight (3 s duration) at the start of a trial. In other words, at the start of each cycle in the signalled component, the houselight flashed on for 0.25s and off for 0.25s and five times in succession. After baseline training, drug testing (i.e., varied doses of d-amphetamine) was conducted similar to experiment 1. Besides attempting to replicate the major result of Experiment 1 - reduction in the sensitivity to delay produced by d-amphetamine - the goals of Experiment 2 were to explore whether reinforcement magnitude affected responding in the rapid-acquisition procedure, and whether drug administration would differentially disrupt responding in the small- and large-magnitude trials.

The first goal relates to an experiment reported by Grace (1999). The purpose of his study was to determine whether pigeons' sensitivity to delay in concurrent chains was affected by the absolute magnitude of reinforcement. Many studies with humans (e.g., Benzion, Rapoport & Yagil, 1989; Green, Fry & Myerson, 1994; Raineri & Rachlin, 1993) found that rate of temporal discounting (i.e., sensitivity to delay) varies inversely with reinforcer magnitude. Grace (1999) found that overall, but not relative, initial-link response rates were affected by reinforcement magnitude. Overall initial-link response

rates were greater with large compared to small magnitude reinforcers, but sensitivity to delay did not vary with reinforcer magnitude. In other words, the amount-dependent discounting that has been obtained with humans, was not obtained with pigeons responding for terminal-link VI schedules in concurrent chains. Other studies have reported a similar lack of effect of reinforcer magnitude on delay sensitivity (e.g., Green et al., 2003; and Richards et al., 1998, with rats; Ong & White, 2004, with pigeons). However, there have been no studies which focus on the effects of overall magnitude on acquisition of choice behaviour.

A possible effect of magnitude on acquisition of choice is suggested by the "signalled magnitude effect" in delayed matching-to-sample (DMTS). Nevin and Grosch (1990) found that rate of forgetting in DMTS was reduced when upcoming large reinforcers for correct responses were signalled. If the concurrent-chains rapid acquisition procedure is viewed as a discrimination task (i.e., choose the shorter terminal-link delay), then it is possible that signalling an upcoming large reinforcer will increase rate of acquisition.

Second, we were interested to explore whether d-amphetamine might act to disrupt responding in a manner consistent with research on resistance to change. According to behavioural momentum theory (Nevin & Grace, 2000), the relationship between reinforcement and behavioural resistance to change can be understood by means of analogy with concepts described in the physics of motion, such as velocity and mass (Nevin & Grace, 2000). On this view, rate of responding is as analogous to velocity and resistance to change of behaviour is as analogous to mass. When a disrupter is imposed, such as suspension of the response–reinforcer contingency (e.g., extinction), decreases in

the value of the reinforcer (e.g., prefeeding food reinforcers) and introduction of alternate sources of reinforcement (Dube et al & McNamara, 2003), responding should decrease relatively less (i.e., be more resistant to change) in the component with the relatively richer conditions of reinforcement (i.e., high rate or large magnitude reinforcer).

There have been several studies which use drugs as disruptors in clinical (e.g. Nevin, 1974, 1979; Nevin & Grace, 2000; Cohen, 1986; Hoffman et al, 1987), although the results have been mixed. For example, Cohen (1986) study was used three different drugs (e.g. d-amphetamine sulfate, sodium pentobarbital, haloperidol, and cholecystokinin-octapeptide) with three multiple chain schedules experiment (e.g. random interval 30 s random interval 30 s; multiple fixed interval 30 s fixed interval 120 s and multiple random interval 30s random interval 120 s) in rats. The results found both cholecystokinin-octapeptide and high doses of d-amphetamine reduce response rate in initial and terminal component; but no difference in rest of components. In other words, Cohen's (1986) results fail to provide evidence that drugs can be viewed as disrupters analogous to prefeeding or response-independent food. Thus, Experiment 2 in present study tested whether initial link responding during the signalled large magnitude component would be more resistant to disruption than in the small-magnitude component.

Method

Subjects:

Four pigeons of mixed breed, numbered 225, 226, 227 and 228, were maintained at 85% plus or minus 15g of their free-feeding weights through appropriate postsession feeding. Pigeons were housed individually in a vivarium with a 12:12 hr light / dark cycle (lights on at 6:00 a.m.), with water and grit freely available in the home cages. Pigeons were experienced with a variety of procedures, but had no prior training with concurrent chains in which delays were changed unpredictably across sessions, as in Experiment 1.

Apparatus:

The apparatus for Experiment 2 was the same as Experiment 1.

Procedure:

The concurrent-chains procedure was similar in most respects to Experiment 1, that is, the initial-link schedule (signaled by white illumination of the side keys) was a VI 10 s that arranged equal entries into the terminal links, which were FI 4 s/FI 16 s on the green key and FI 8 s on the red key. All other details were the same as Experiment 1, with the following exceptions. First, the terminal-link assignments were not counterbalanced across pigeons. For all pigeons, the FI 8-s terminal link was signalled by red illumination of the left key, and the FI 4 s/FI 16 s terminal link was signalled by green illumination of the right key. The second difference was that two types of concurrent-chains cycles were included, in which the reinforcers for the left and right terminal links were either both small magnitude (1.5 s access to food) or both large magnitude (4.5 s access to food). Trial-types were signalled by presence (or absence) of a flashing houselight (3 s duration) at the start of a trial. In other words, at the start of

each cycle in the signalled component, the houselight flashed off for 0.25s and on for 0.25s, for a total of 3 s. Sessions consisted of 72 cycles, which comprised 12 blocks of 6 trials. All trials were the same (flashing/not flashing) within each block. The identity of the first block was determined randomly, and strictly alternated thereafter. For Pigeons 225 and 226, large magnitude reinforcers were delivered during the signalled component, and small magnitude reinforcers during the unsignalled (i.e., no flashing houselight at the beginning of trials) component. For Pigeons 227 and 228, these assignments were reversed. As in Experiment 1, sessions ended after 70 minutes if 72 cycles had not yet been completed.

Once pigeons' responding stabilized after 31 sessions training in the baseline, drug testing began during the second PRBS. Drug testing was similar to Experiment 1; during drug testing varied doses of d-amphetamine; 0.3, 1.0, 1.7 and 3.0 mg/kg were given. Injections were administered once or twice week, provided that the data from the session conducted the day before (the control session) was within the range of the previous 10 non-injection sessions. The drug injections were the same as Experiment 1, that is, each dose was given in pairs, once at FI 4 s and once at FI 16 s before testing the next dose. Once each dose had been tested in pair, doses of 1.0 and 1.7 mg/kg were the probed in additional sessions (Table 3).

Pigeon 225	**Pigeon 226**
Saline—Three doses for 4s delay Three doses for 16s delay. 0.3 mg/kg—Two doses for 4s delay One doses for 16s delay. 1.0 mg/kg—Three doses for 4s delay Two doses for 16s delay. 1.7 mg/kg—Two doses for 4s delay Three doses for 16s delay. 3.0 mg/kg—One doses for 4s delay One doses for 16s delay.	Saline—Three doses for 4s delay Three doses for 16s delay. 0.3 mg/kg—Two doses for 4s delay One doses for 16s delay. 2.0 mg/kg—Three doses for 4s delay Two doses for 16s delay. 1.7 mg/kg—Two doses for 4s delay Three doses for 16s delay. 3.0 mg/kg—One doses for 4s delay One doses for 16s delay.
Pigeon 227	**Pigeon 228**
Saline—Three doses for 4s delay Three doses for 16s delay. 0.3 mg/kg—Two doses for 4s delay One doses for 16s delay. 3.0 mg/kg—Three doses for 4s delay Two doses for 16s delay. 1.7 mg/kg—Two doses for 4s delay Three doses for 16s delay. 3.0 mg/kg—One doses for 4s delay One doses for 16s delay.	Saline—Three doses for 4s delay Three doses for 16s delay. 0.3 mg/kg—Two doses for 4s delay One doses for 16s delay. 4.0 mg/kg—Three doses for 4s delay Two doses for 16s delay. 1.7 mg/kg—Two doses for 4s delay Three doses for 16s delay. 3.0 mg/kg—One doses for 4s delay One doses for 16s delay.

Table 3: Doses of the drug administration (d-amphetamine) were used for Pigeons 225 through 228 in present study.

Results and Discussion

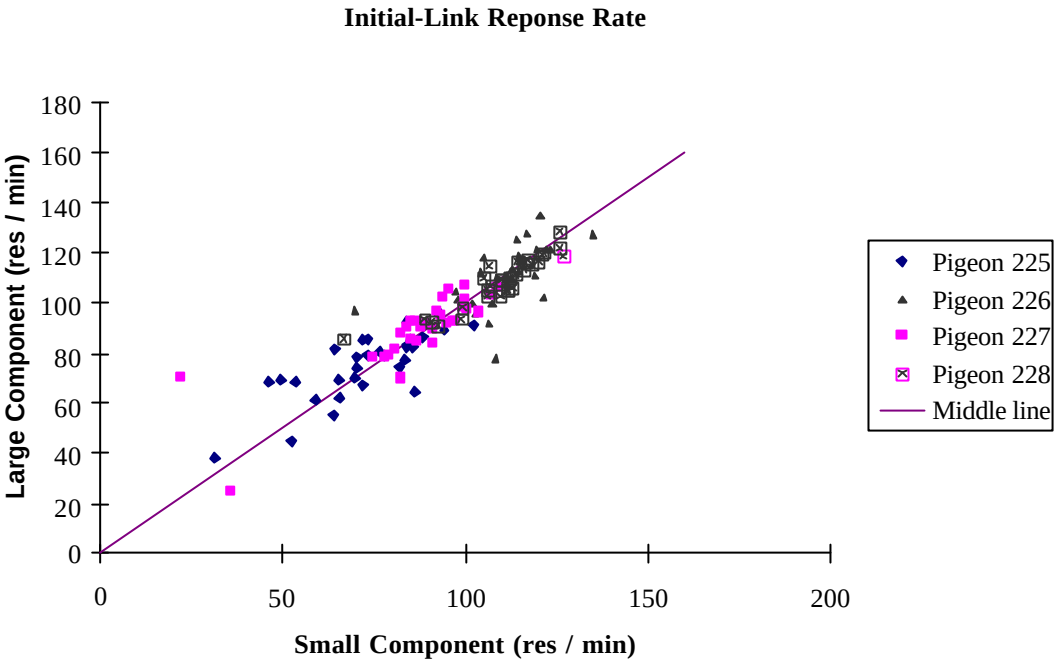


Figure 18: The overall initial-link response rate in the larger magnitude component as a function of the corresponding rate in the small magnitude component in baseline condition. Data were pooled across the last 31 sessions of baseline prior to drug testing. The major diagonal (line of equality) is also shown.

Baseline data

Data from the last 31 sessions of baseline were analyzed. The first question was whether reinforcer magnitude affected overall initial- or terminal-link responding. Figure 18 shows, for all subjects, the overall initial-link response rate in the large magnitude component (y-axis) plotted against overall initial-link response rate in the small magnitude component (x-axis). Overall initial-link response rate was calculated as the sum of responses made to both initial links divided by the total time spent responding

in the initial links. It is clear that data points in Figure 18 scatter unsystematically around the major diagonal, which indicates equal response rates. This indicates that for all subjects, there were no systematic differences in overall initial-link response rate between large magnitude and small magnitude components. This result is contrary to that reported by Grace (1999), who found that overall initial-link response rate was greater in the large magnitude component.

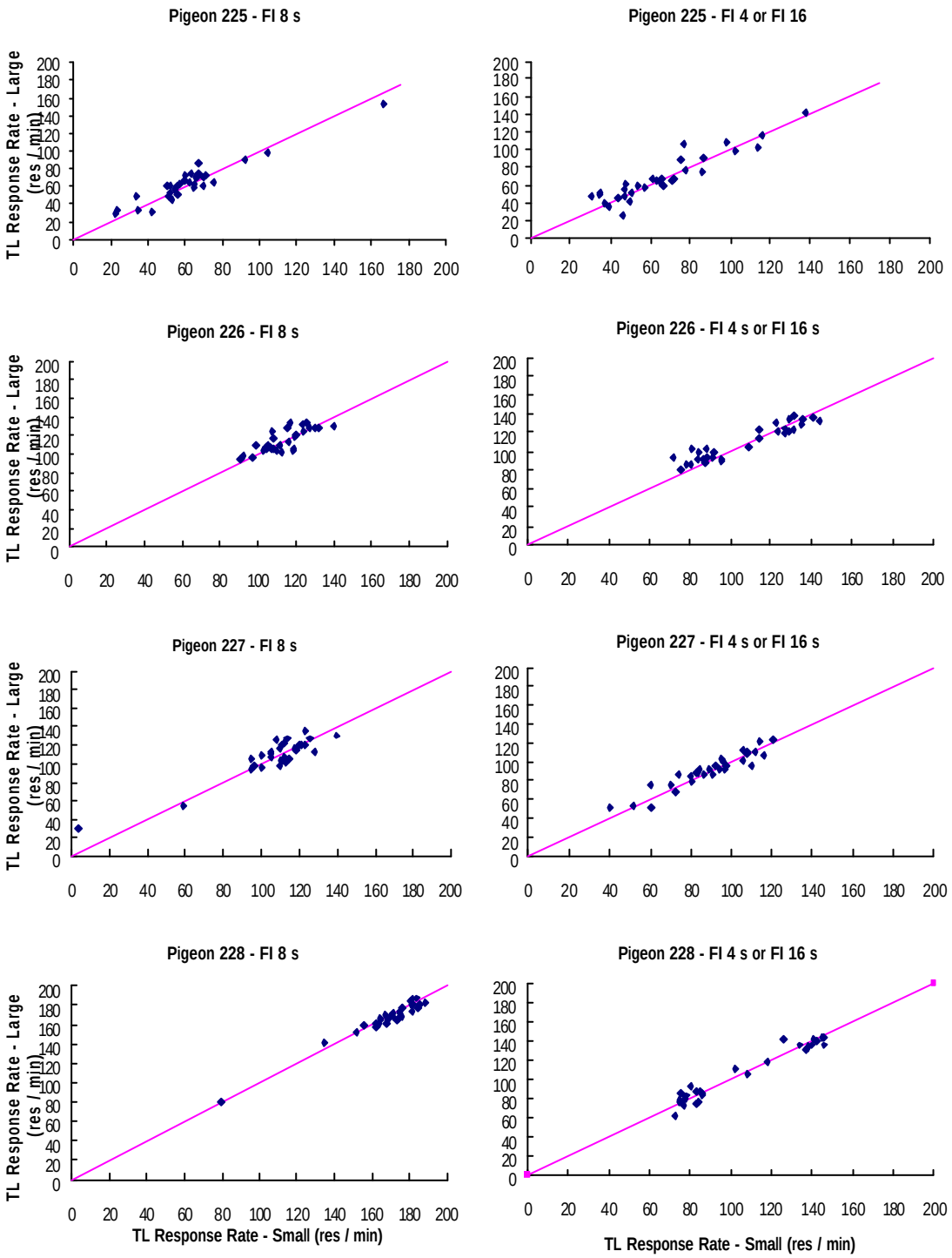


Figure 19: The terminal link response rate in the larger magnitude component as a function of the corresponding rate in the small magnitude component. Data from the FI 8-s terminal link are shown in the left panels; data from the FI 4-s / FI 16-s terminal link are shown in the right panels. Data were aggregated across the last 31 sessions of baseline prior to drug testing. The major diagonal (line of equality) is also shown.

Corresponding terminal-link data are presented in Figure 19, which shows the terminal link response rate in the large magnitude component plotted as a function of the terminal-link response rate in the small magnitude component. Similar to the initial-link data above, data points for all schedules and subjects are unsystematically scattered around the major diagonal. This indicates that there is no systematic difference in terminal-link response rate depending on whether the magnitude of the upcoming reinforcer was large or small. This result is similar to that reported by Grace (1999). Thus, there were no effects of reinforcer magnitude on overall initial- or terminal-link responding in the present study.

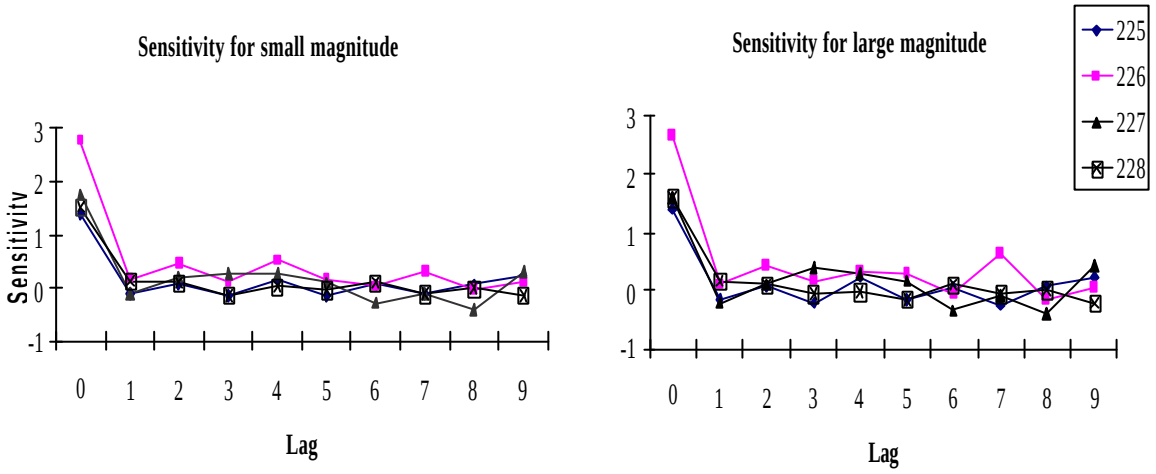


Figure 20: Sensitivity of the small and larger magnitude in Lag 0 through Lag 9 for all subjects within session.

Figure 20 shows Lag 0 through Lag 9 sensitivity coefficients for responding in the small and large magnitude components, for all subjects. All subjects are accounting variety between 83% to 94% variance for small magnitude components e.g. Pigeon 225, 93%; Pigeon 226, 94%; Pigeon 227, 89% and Pigeon 228, 83% and accounting variety between 89% to 93% variance for large magnitude components e.g. Pigeon 225, 92%;

Pigeon 226, 93%; Pigeon 227 and Pigeon 228, 89%.The regression coefficients for pigeon 225 shows significant result for Lag 0 in small and large magnitude (Lag 0 sensitivity=1.38 for small magnitude and sensitivity=1.40 for large magnitude, $p<0.05$). There is a Type one error for Lag 4, Lag 7 and Lag 9 for small or large magnitude. The regression coefficients for pigeon 226 showed a significant result for Lag 0 in small and large magnitude (Lag 0 sensitivity=2.74 for small magnitude and sensitivity=2.66 for large magnitude, $p<0.05$). There is a type one error for Lag 2, Lag 4 and Lag 7 for small or large magnitude. The regression coefficients for pigeon 227 showed a significant result for Lag 0 in small and large magnitude (Lag 0 sensitivity=1.77 for small magnitude and sensitivity=1.58 for large magnitude, $p<0.05$). There is a type one error for Lag 3, Lag 6, Lag 8 and Lag 9 for small or large magnitude. The regression coefficients for pigeon 228 shows significant result for Lag 0 in small and large magnitude (Lag 0 sensitivity=1.53 for small magnitude and sensitivity=1.58 for large magnitude, $p<0.05$) whereas coefficients for rest of Lag were never significant (varied from -0.14to 0.16). There is no type one error from Lag 1 through Lag 9 for small or large magnitude. These results replicate Experiment 1 in showing that pigeons' response allocation was sensitive to the immediacy ratio in the current session. However, there were no systematic differences in Lag 0 sensitivity between small and large magnitude components (e.g. the average of Lag 0 sensitivity for small magnitude component is 1.85 and the average of Lag 0 sensitivity for large magnitude component is 1.81).

Summary for baseline condition

There were no systematic differences between small and large magnitude for either initial link and terminal link response (Figure 14 and Figure 15). There are also no systematically differences between small and large magnitude for sensitivity within session. Overall, the present study found similar results as Grace (1999) study in terms of sensitivity and terminal-link response rates, but failed to obtain the difference in overall initial-link response rate reported by Grace (1999).

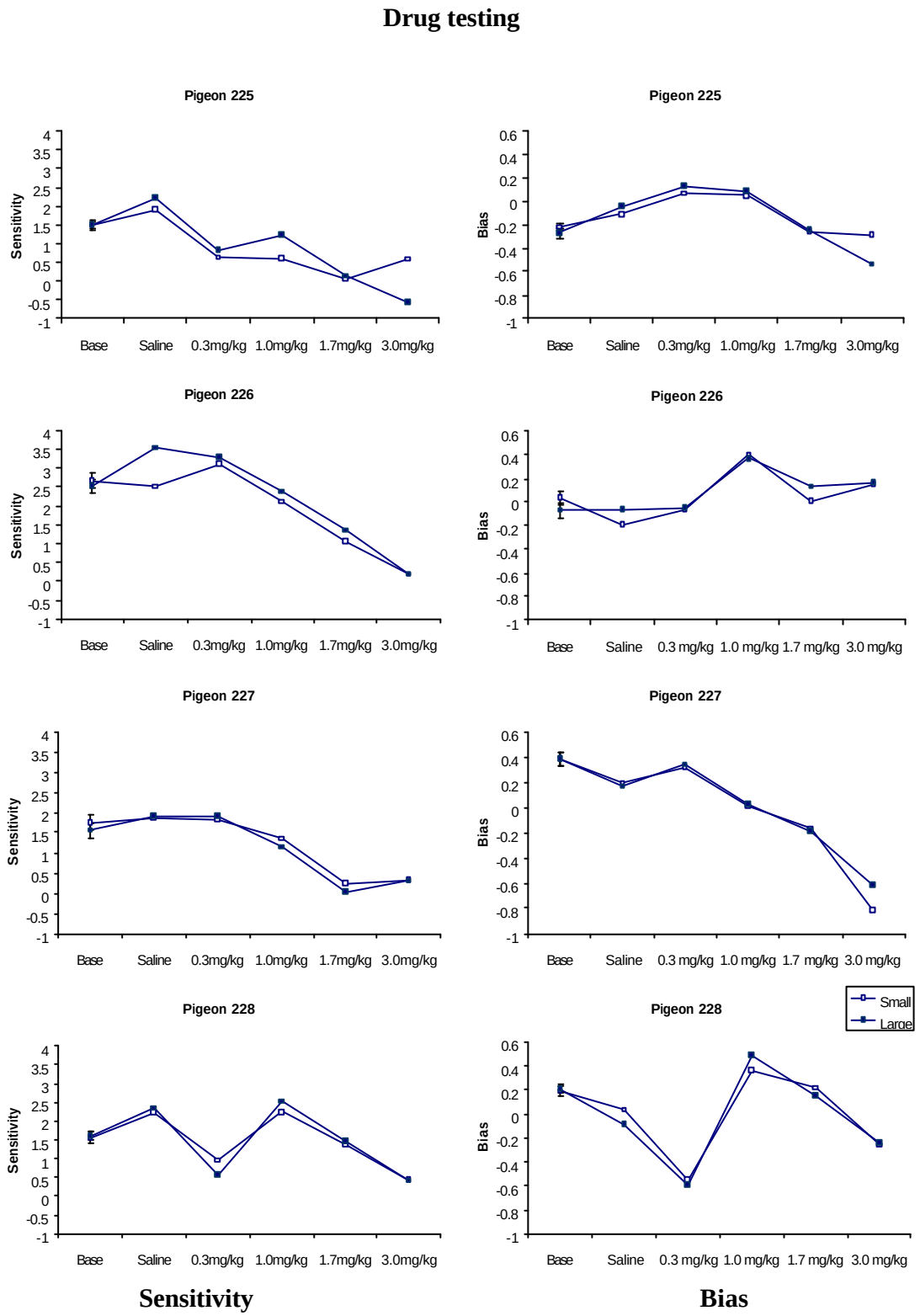


Figure 21: Sensitivity and bias in the small and large magnitude for drug condition for all subjects. Error bars indicate plus or minus one standard error.

As in Experiment 1, data were pooled across multiple determinations of a drug dose with a particular set of terminal-link schedules. Point estimates of bias and sensitivity (Lag 0) were calculated, separately for the large and small magnitude components. Results are shown in Figure 21, which also includes baseline data for sake of comparison.

Data for Pigeons 225, 226 and 227 (left-hand panels) show that sensitivity decreased monotonically (or nearly so) when amount of d-amphetamine was increased in both small and large magnitude components. There was a slight difference between small and large magnitude components in sensitivity with different amount of d-amphetamine for Pigeon 225 but almost no differences for Pigeon 226 and 227. Data for Pigeon 228 were more variable - sensitivity decreased from baseline through 0.3 mg/kg but increased in 1.0 mg/kg, and decreased again through 3.0 mg/kg. However, with the exception of 1.0 mg/kg, sensitivity values during drug sessions were always lower than baseline. Again, there was almost no difference between small and large magnitude in sensitivity with different amount of d-amphetamine for Pigeon 228.

Pigeon 225 showed a slight right key bias (right-hand panels) at beginning of the baseline condition with large and small magnitude components, but this was eliminated when given doses of 0.3 and 1.0 mg/kg of d-amphetamine. However, pigeon 225 developed right key bias again when given doses of 1.7 and 3.0 mg/kg of d-amphetamine. There was almost no difference between large and small magnitude components for bias for pigeon 225. Pigeon 226 shows almost no bias for large and small magnitude components in the beginning of the baseline condition but developed slightly left key bias when given doses of 1.0 mg/kg of d-amphetamine. However, the left key bias was

eliminated when given doses of 1.7 and 3.0 mg/kg of d-amphetamine. There was almost no difference between large and small magnitude components for bias for pigeon 226. Pigeon 227 showed a left key bias at beginning of the baseline condition but eliminated when given doses of saline, 0.3 and 1.0 mg/kg of d-amphetamine. However, this pigeon developed a right key bias when given 1.7 and 3.0 mg/kg of d-amphetamine. Again, there was no difference between large and small magnitude components. Pigeon 228 shows left key bias at beginning of the baseline condition but it was eliminated in saline test sessions. However, responding in both large and small magnitude components developed a strong right key bias for doses of 0.3 mg/kg and a strong left key bias for doses of 1.0 mg/kg. These biases were eliminated when given doses of 1.7 and 3.0 mg/kg of d-amphetamine. Once again, there were no differences in bias between large and small magnitude components. Overall, results in Figure 21 replicate those obtained in Experiment 1: increasing dosage of amphetamine reduced sensitivity to the immediacy ratio in the current session, and bias did not change systematically across subjects. This reduction in sensitivity (and unsystematic changes in bias) was similar for both small and large magnitude components, suggesting that reinforcer magnitude had virtually no effect on response allocation.

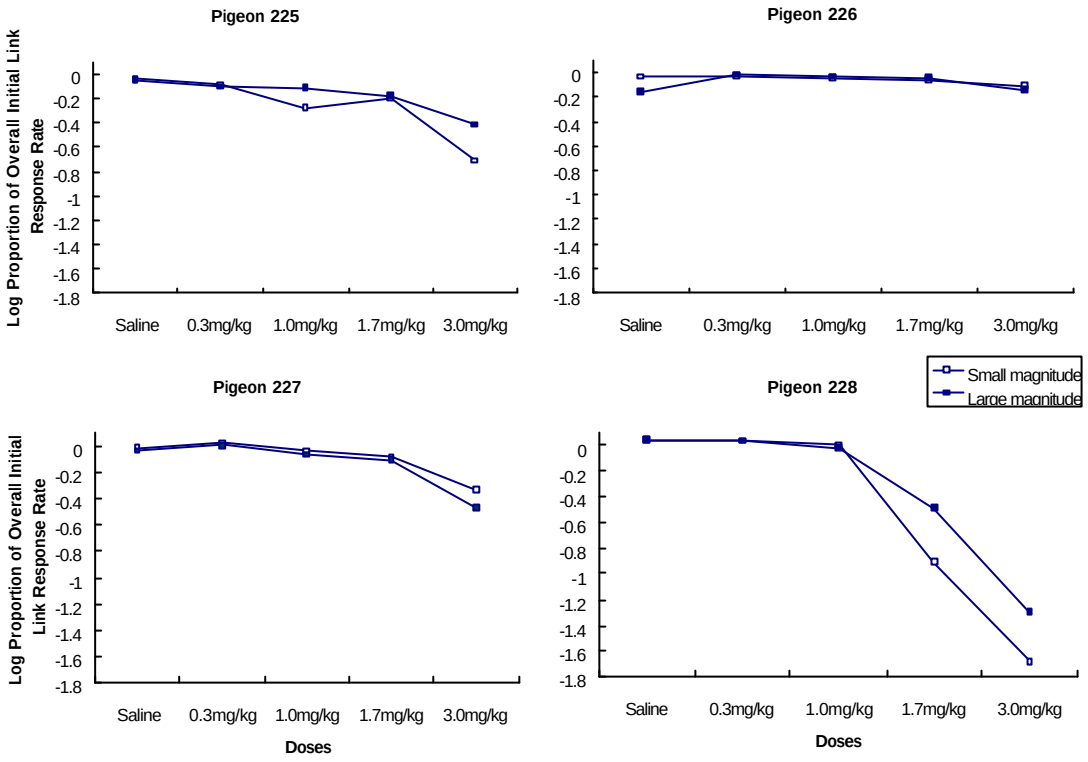


Figure 22: Resistance to change in the drugs conditio n at the initial link response rate for all subjects. Resistance to change was measured as the log proportion of overall initial-link response rate in baseline.

Finally, we were interested to test whether administration of amphetamine would differentially disrupt overall initial-link responding in the large- and small-magnitude components. According to behavioral momentum theory (Nevin & Grace, 2000), responding should be more resistant to change in the large magnitude component. Figure 22 shows, for all subjects, overall initial-link response rate as a log proportion of baseline response rate during test sessions, separately for the large and small magnitude components (indicated by filled and unfilled squares, respectively). For all subjects, responding decreased with increasing doses of amphetamine, indicating that drug administration disrupted responding. Pigeon 225 and 228 shows responding decreased

relatively more rapidly in the small magnitude component rather than larger magnitude when increased the amount of d-amphetamine. Pigeon 227 shows that responding slightly decreased relatively more in the large magnitude component than small magnitude when increased the amount of d-amphetamine. Pigeon 226 shows no systemically difference for large and small magnitude component when increased the amount of d-amphetamine. Thus, overall initial-link responding decreased as the amphetamine dose increased, but there were no systematic differences between the large- and small-magnitude components. Responding in the large magnitude component was slightly more resistant to change for Pigeon 225 and 228, but the opposite was obtained for Pigeon 227, and there was no systematic difference for Pigeon 226.

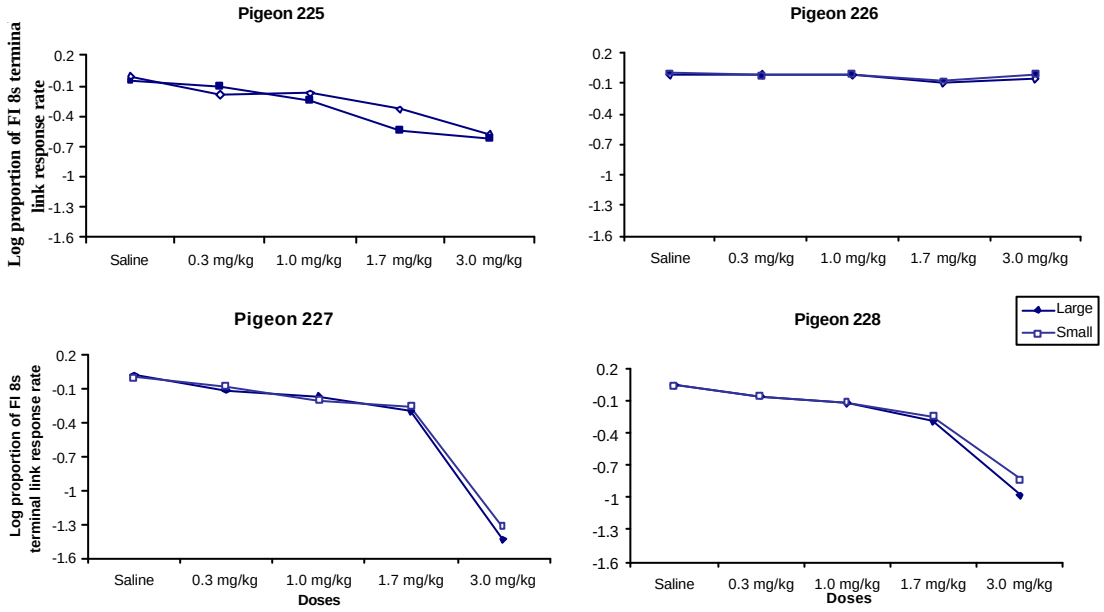


Figure 23: Resistance to change in the drugs condition for responding during the FI 8-s terminal link for all subjects. Resistance to change was measured as the log proportion of FI 8-s terminal link response rate in baseline

Figure 23 shows, for all subjects, FI 8s terminal link response rate as a log proportion of baseline response rate during test sessions, separately for the large and small magnitude components (indicated by filled and unfilled squares, respectively). The reason for only showing the FI 8-s terminal link in Figure 19 (rather than FI 4 s or 16 s) is that the FI 8-s was presented for all drug sessions, and thus provides the best test of whether drug administration differentially affected terminal-link responding. With the exception of Pigeon 226, responding decreased with increasing doses of amphetamine, indicating that drug administration disrupted terminal-link responding. For the remaining subjects (Pigeon 225, 227 and 228), responding decreased during test but at approximately the same rate in both the small- and large-magnitude component. Overall, therefore, there were no systematic differences in resistance to change between small and large magnitude component for all subjects.

According the literature, terminal-link responding is expected to be more resistant to change than initial link responding (Nevin et al, 1981). Pigeon 225 shows slightly less resistance to change in the terminal link in the large magnitude component (averaging across drug test sessions, -0.18 and -0.32 for initial and terminal-link responding, respectively) but almost the same resistance to change in the small magnitude component between initial link and terminal link responding (i.e., -0.27 and -0.26). Pigeon 226 shows almost the same resistance to change in both small and large magnitude component with both initial link and terminal link response (i.e. -0.09 and -0.02 for small magnitude; -0.06 and -0.04 for large magnitude). Pigeon 227 showed less resistance to change in the terminal link in both large and small magnitude (i.e., -0.09 and -0.40 for small magnitude and -0.13 and -0.40 for large magnitude). Pigeon 228 showed more

resistance to change in the terminal link in both large and small magnitude (i.e., -0.50 and -0.24 for small magnitude and -0.35 and -0.28 for large magnitude). Overall, compare both initial link and terminal link response found that terminal link shows slightly less resistance to change, averaged across subjects, than initial link responding (i.e., -0.24 for terminal link with average of small and large magnitude and -0.21 for initial link with average of small and larger magnitude). Thus, the present study failed to confirm that terminal link responding was more resistance to change than initial-link responding.

The failure to find a difference in terms of resistance to change, together with the lack of any differences between the large and small magnitude components in terms of overall and relative initial- and terminal-link responding in baseline, and bias and sensitivity during drug testing, suggests that the reinforcer magnitude manipulation was ineffective. Perhaps signaling the presence of a large (or small) reinforcer magnitude briefly at the start of an initial-link cycle is insufficient to generate stimulus control over responding.

Summary for drug condition:

All subjects showed decreases in sensitivity when given increased doses of d-amphetamine (Figure 21), but bias did not change systematically. This replicates results of Experiment 1. Null results were found in resistance to change compared with literature, that is, there was no difference in resistance to change of initial-link and terminal-link responding in the small and large magnitude components (Figure 22 and 23). Terminal link shows less resistance to change than initial link. In other words,

increasing doses of d-amphetamine would not differentially disrupt overall initial-link and terminal-link responding in the large- and small-magnitude components.

General Discussion

The present research demonstrates that the generalized matching model provided an excellent fit to the data for all subjects responding in concurrent-chain schedules in which the terminal-link immediacy ratio changed unpredictably according to a pseudo-random binary sequence (PRBS). However, for all subjects sensitivity to the immediacy ratio decreased when given increasing doses of d-amphetamine. This result suggests that amphetamine affects overall sensitivity within sessions. There were no systematic differences between small and large magnitude for either initial link and terminal link response with baseline and drug condition in Experiment 2. There was also no difference in resistance to change of initial-link responding in the small and large magnitude components in drug condition. Those results indicated that reinforcement magnitude did not affect responding in the rapid-acquisition procedure, and the drugs administration did not differentially disrupt responding in the small- and large-magnitude trials. In addition, the present study fails to show that terminal-link responding was more resistant to change than initial-link responding (Nevin et al., 1981).

In the baseline condition of Experiment 1, the red-key schedule was always FI 8 s while the green-key schedule varied between either FI 4 s or FI 16 s across sessions according to a 31-step PRBS. Response allocation for all subject successfully tracked unpredictable changes in the immediacy ratio. This result was similar to Grace et al (2003) study expect the sensitivity value was higher in present study. In the Grace et al's (2003), the sensitivity to immediacy ratio (e.g. Lag 0) was 1.04 on average. In present study, the sensitivity to immediacy ratio was 1.33 on average. The present study also has higher sensitivity for Lag 0 in sixth of the session in average than in Grace et al's study

(e.g. 1.49 for present study and 1.37 for Grace et al's study). One explanation for the differential sensitivity could be due to differences between the studies in the total amount of training. Normally, sensitivity would be expected to increase with larges amounts of training. However, in Experiment 1 of the present study, subjects received less than total amount of training in Grace et al's (2003) study (e.g. 74 training in present study and 93 training in Grace et al's study). Nevertheless the sensitivity was higher in present study than Grace et al's (2003) study. Given that there were individual differences in sensitivity within each study, the present results constitute a fairly close replication of Grace et al. (2003), both in terms of the average sensitivity to immediacy and also the degree of individual differences. The generalized-matching model (Equation 8) described the data from the present study very well, accounting for an average of 97% of the variance in response allocation. This compares favorably to Grace et al.'s (2003) study (94%), as well as Schofield and Davison's (1997) study with concurrent schedules (95%).

We also found, similar to Grace et al. (2003), that response allocation changed to a greater extent within session when the green-key delay was 16 s rather than 4 s. As Grace et al. (2003) noted, this result is similar to the well-known "terminal link effect" in steady-state concurrent chains research, that is, the preference between a pair of schedules in constant ratio (2:1) becomes more extreme as their absolute duration increases (Grace & Bragason, 2004; MacEwen, 1972; Williams & Fantino, 1978). Although this finding is inconsistent with simple generalized-matching models such as Equation 8, it is predicted by more complex models for concurrent chains, based on the

generalized matching law, such as Grace's (1994) contextual choice model and Mazur's (2001) hyperbolic value-added model.

Drug test sessions were conducted after baseline training in Experiment 1, and subjects were injected with varying doses of d-amphetamine (e.g. saline, 0.3, 1.0, 1.7 and 3.0 mg/kg), 15 minutes before session start and once or twice a week. For all subjects, sensitivity to the current-session immediacy ratio decreased as the dose of d-amphetamine was increased. This result supports the basic conclusion of Pitts and Febbo (2004) study, that is, the sensitivity to delay will decrease when the dose of methamphetamine increased. One explanation suggested by Pitts and Febbo (2004) was that methamphetamine administration attenuated the discounting effects of reinforcement delay to organism. The sensitivity change can be also described in terms of behavioral mechanisms of drug action theory and rate-dependency theory. In behavioral mechanisms of drug action theory, that is, understanding how environmental contingencies modulate the behavioral effects of drugs, explained d-amphetamine could change the organism's capacity to execute the response; the effects of the establishing operation (e.g., d-amphetamine will suppress the appetite), the effects of the contingency between behavior and the consequent events (e.g., environmental history can modulate the behavioral of the drug action and change the sensitivity to delay), and the nature of the antecedent stimulus control (e.g., food deprivation and modulation of behavior regulated by aversive stimulation could alter the efficacy of drug and change sensitivity to delay; Pitts & Febbo, 2004; Thompson, 1984; Branch, 1984, 1991; Thompson & Schuster, 1968). According to rate-dependency theory, which maintains that how drugs change the rate of responding depends on response rate in baseline, explained that the

novelty effects of drugs could cause behavioral changes and make change for sensitivity to delay. Specifically, if amphetamine reduces response rate to a larger extent when response rates are high compared to when they are low, then sensitivity would be reduced. This would be independent of any specific pharmacological actions that the drug might have (Dews, 1962; Thompson & Schuster, 1968). Overall, both behavioral mechanisms of drug action theory and rate-dependency theory can describe the relationship between sensitivity changes with different amount of drugs in present study.

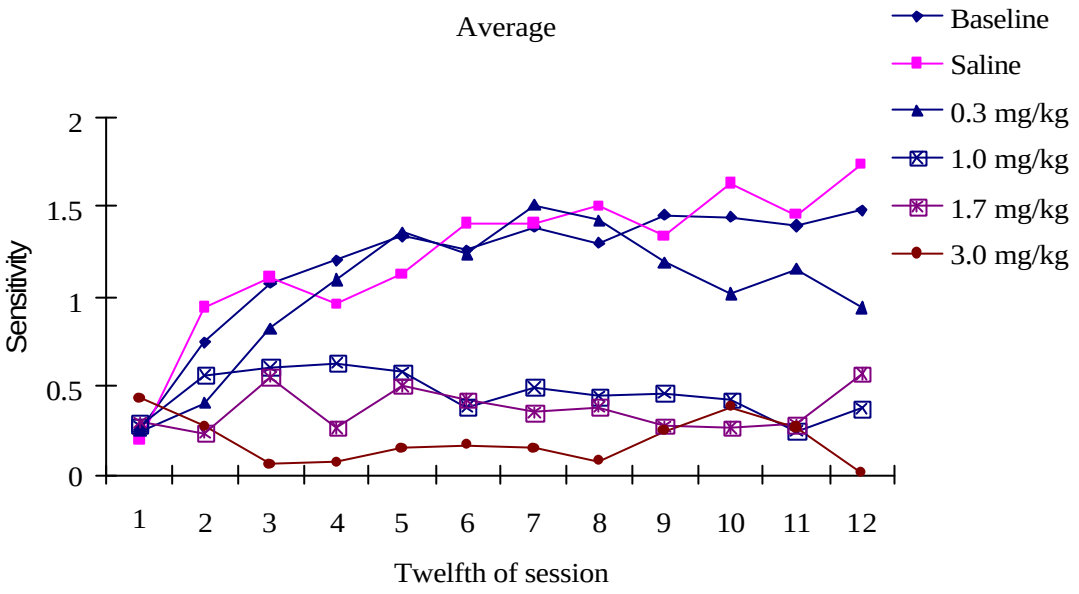


Figure 24: Average sensitivity in twelfth of session for all subjects in Experiment 1.

If amphetamine reduces sensitivity overall, then an interesting question becomes whether it affects the rate of change within sessions. Although variability in the individual data in Experiment 1 made this question difficult to address, some insight may perhaps be gained from the average data. Figure 24 shows sensitivity to the current-session immediacy ratio for eachsession twelfth, averaged across all subjects in Experiment 1. Figure 24 shows that the sensitivity was reduced at the intermediate and

higher dose levels, but it did not change over the course of the session. There was almost the same pattern between baseline and saline. There was some evidence of reduction in sensitivity compared to baseline condition for lower doses of d-amphetamine (e.g. 0.3 mg/kg), but the rate of acquisition appeared to be approximately the same as baseline condition. However, for intermediate and higher doses of d-amphetamine (e.g. 1.0, 1.7 and 3.0 mg/kg) asymptotic sensitivity (i.e., in the second half of the session) was reduced further, yet there were no systematic changes within the session. Therefore, although amphetamine may attenuate sensitivity, there is no evidence from the present research that it attenuates the rate of learning within session. Given a sufficient dose of amphetamine, the rate of change in preference was reduced effectively to zero.

Many studies have found that the errors increase and response rate decreases in repeated acquisition, or the likelihood of choosing a larger, more delayed reinforcer increases with higher doses of d-amphetamine for pigeons (Thompson & Moerschbaecher, 1979; Harting & McMillan, 1976; Richards et al, 1999; Pitts & Febbo, 2004). The results for present study are consistent with the response rate decreases with higher doses of d-amphetamine for pigeons, and also suggest that pigeons will be more likely to choose a larger, more delayed reinforcer with higher doses of d-amphetamine. Considering the wider implications of results, there are potential connections to human studies. For example, moderate doses of d-amphetamine can decrease impulsivity and improve attention and learning in children who have suffered with attention deficit hyperactivity disorder (ADHD; Gillberg et al, 1997; Findling and Dogin, 1998; Solanto, 1998). In other word, d-amphetamine can reduce the sensitivity to delay in self-control procedure with children diagnosed with ADHD. This result is similar to present research

that d-amphetamine reduced the sensitivity to delay in pigeons. By contrast, drug abusers who chronically use amphetamine, showed an increase in impulsivity and long-term impairments in learning and attention (Richards et al, 1999). The same result was found with smokers, alcohol users, and opium and heroin users (Richards et al, 1999; Bickel et al, 1999; Kirby et al, 1999). In other word, those studies imply that chronic drug uses will have increased sensitivity to delay (i.e., be more impulsive). This may appear to conflict with the results of the present research, which found that d-amphetamine reduced the sensitivity to delay. However, the cause or effect relationship is unclear, because individuals who are more impulsive may be more likely to become drug abusers. Secondly, the sizes of doses used by chronic drug abusers and in treatment for ADHD vary.

Results of Experiment 2 provided further support for these conclusions. Experiment 2 was similar to Experiment 1, except that the overall reinforcer magnitude differed across trials. The goals of Experiment 2 were to explore whether reinforcement magnitude affected responding in the rapid-acquisition procedure, and whether drug administration would differentially disrupt responding in the small- and large-magnitude trials. In the baseline condition, results were similar to Experiment 1; that is, pigeons' response allocation was sensitive to the immediacy ratio in the current session for both small and large magnitude components. However, there were no systematic differences in Lag 0 sensitivity between small and large magnitude components within the session. The generalized matching model (Equation 8) also provided an excellent fit in both small and large magnitude components in Experiment 2 (94% for small magnitude component and 90% for large magnitude component). However, there were no effects of reinforcer

magnitude on overall initial- or terminal-link responding in baseline condition at present study. In other word, amount-dependent temporal discounting was not found responding in both initial-link and terminal-link with VI schedules in concurrent chains for pigeons. In addition, there were no systematic differences between small and large magnitude components in bias for all subjects. Similar results were found by Grace (1999) study, that is, relative initial link response rates were not systematically different in the small and large magnitude components. In other words, there was no evidence that the sensitivity to delay was affected by magnitude.

Many studies have found evidence for amount-dependent temporal discounting in humans, that is, humans will discount a larger amount of money at lower rates than a smaller amount of money (e.g., Benzion, Rapoport & Yagil, 1989; Green, Fry & Myerson, 1994; Raineri & Rachlin, 1993). If sensitivity to immediacy is regarded as a measure of temporal discounting, then results of Experiment 2 failed to show evidence of amount–dependent temporal discounting. The failure to find an effect on overall initial-link response rate in the present research could be due to an ineffective manipulation. For example, during the signaled component, the flashing houselight might need to signal longer than 3 s or signal throughout entire initial link schedule. Apparently, the 3-s flashing houselight failed to gain control over behavior, but a longer signal could eliminate this problem. Use of different colored flashing houselight for the different components (e.g. yellow flashing houselight for small magnitude component and blue flashing houselight for large magnitude component), could make the components more distinguishable to pigeons. Because the present research failed to find evidence of the "signaled magnitude effect", that is, that the level of performance was higher in the

signals for large reinforcers than for the small reinforcers (Nevin & Grosch, 1990), it is possible that acquisition may be faster on signalled larger reinforcer trials, given effective stimulus control. Therefore, the negative results from present research should not be taken to mean that overall magnitude does not affect acquisition in a PRBS design with concurrent-chain schedules.

Results of drug testing in Experiment 2 also confirmed the results in Experiment 1, that is, sensitivity was decreased when doses of d-amphetamine were increased in both small and large magnitude components. However, the reinforcer magnitude had virtually no effect on response allocation with drugs condition. In addition, no systematic differences were found in terms of resistance to change of initial-link responding in the small and large magnitude components. In other words, the present research failed to confirm predictions of behavioral momentum theory. According to behavioral momentum theory, responding that is more richly reinforced will be relatively more resistant to change when a disruptor such as prefeeding, extinction, drugs or response-independent food is employed (Nevin & Grace, 2000). Because the flashing houselight in the present study may not have acquired effective control, it is unclear whether the negative results for resistance to change of initial-link responding are evidence that d-amphetamine cannot be viewed as a disruptor similar to those mentioned above (cf. Cohen, 1986).

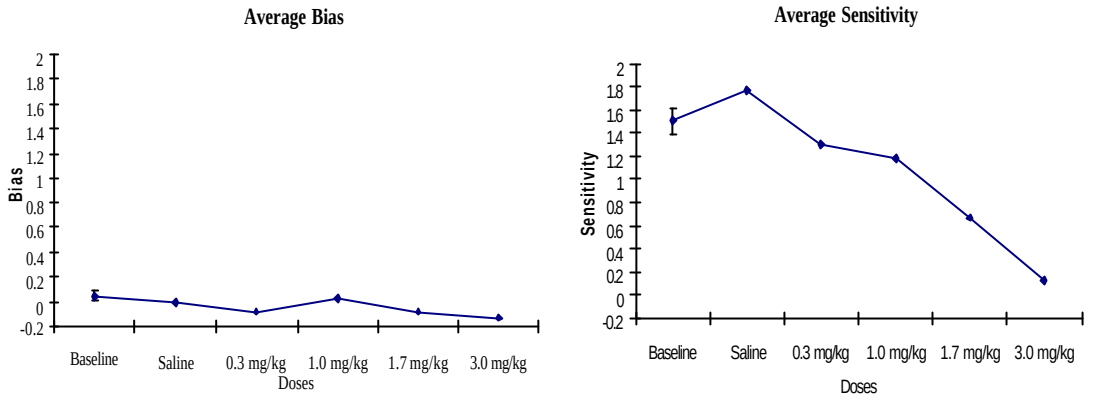


Figure 25: Average bias and sensitivity across Experiment 1 and Experiment 2 for eight subjects.

Figure 25 shows the average bias and sensitivity across two experiments for all subjects in present research. Clearly, there were no systematic differences between bias with different doses of d-amphetamine for eight pigeons in two experiments. The standard deviation varied between 0.05 to 0.10 for average bias. Figure 24 also gives strong evidence that sensitivity was decreasing when increased the doses of d-amphetamine for both experiments. The standard deviation varied between 0.06 to 0.32 for average sensitivity.

In summary, the present research results suggest that pigeons have ability to adjust response allocation rapidly when given unpredictable changes in the terminal-link FI schedule across sessions. Sensitivity to the current-session immediacy ratio decreased when pigeons were injected with increasing doses of d-amphetamine for both experiments, and this finding can be explained both in terms of behavioral mechanisms of drug action theory and rate-dependency theory. Experiment 2 provided no evidence that reinforcement magnitude affected initial-link responding in the rapid-acquisition procedure. There was also no evidence that d-amphetamine differentially disrupted

responding in the small- and large-magnitude trials, contrary to predictions of behavioural momentum theory.

There are some limitations in the present research. In some cases, the drugs data were variable. For example, subjects sometimes did not complete all the trials during drugs sessions, and ceased responding with higher doses. Additional drug testing could be conducted to obtain more reliable data in present research. However, it seems unlikely that these data would be systematically different from the present results. Future extensions could be to use multiple concurrent-chain schedules (e.g. design more than two alternative keys in concurrent-chain schedule with various terminal link schedules) to find out how rapidly the response allocation can adjust to the frequent changes in the terminal links. Using varied drugs (e.g. cocaine and heroin) to testing how the sensitivity change and find out what are the difference compare with d-amphetamine in concurrent-chain schedule. Using varied drugs could also find out whether rate of learning within the session can be affected by other drugs. In addition, a more effective manipulation to signal reinforcer magnitude in Experiment 2 could be used, such as using different colored flashing houselights, or different colored initial-link stimuli.

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